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CANCER
ITS
USE AND TREATMENT
—
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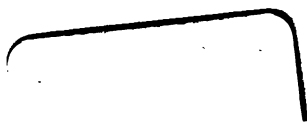
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PAUL B. HOEBER, 67-69 East 59th St., N. Y.

CANCER

ITS

CAUSE AND TREATMENT

BY
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To
THE GOVERNORS
of the
NEW YORK SKIN AND CANCER HOSPITAL
whose kind appreciation of and assistance to the author
in his clinical work in their institution have
done much to encourage him and to
promote the interest of the pro-
fession in the branch of
DERMATOLOGY
this little book is inscribed



PREFACE

CANCER has hitherto been regarded almost wholly from its histological and surgical aspects. The investigators and practitioners along these lines have been innumerable, the work done prodigious, and the literature relating thereto enormous. But relatively little attention has been paid to the medical aspects of this most threatening malady, although voices have been raised from time to time, with more or less force, all joining in the same plea that the basic cause of the disease lies in some derangement in the vital forces of the organism, as influenced largely by diet and mode of life.

It has, therefore, seemed to the writer that it was time to stop and look at cancer from a broader aspect, and by synthesis

and deduction to seek to understand some of the underlying causes of malignant disease; also to see if some general principles could not be ascertained upon which could be based an explanation of the views he had long held, and of the practice which he had long followed in regard to the prevention and cure of this dire malady. And as his studies progressed he was more and more satisfied that the true solution of the cancer problem lay along the lines indicated, although there was still very much to be learned regarding the details and application of the principles involved.

The lectures were prepared for and delivered to practicing physicians at the New York Skin and Cancer Hospital, in connection with the regular Wednesday lectures on Diseases of the Skin, as I felt that I should give them the benefit of what I felt to be of value in my own practice, private and public.

Although holding the views here ex-

pressed for many years I have hesitated writing strongly on the subject before, lest I should be misunderstood or misjudged: for favorable results based solely on a clinical diagnosis of cancer are always doubted. I have also feared lest by advocating a dietetic and medical consideration and treatment of cancer, which possibly might not be carried out exactly, I might really do harm; since some might be led to neglect operative measures in proper cases, at the proper time; and so in certain instances great injury and injustice might be done to the patient, and the time pass in which a surgical operation might possibly be of service.

But with my views and experience, strengthened greatly by what studies I have been able to make in the midst of a busy professional life, I now feel that I should do wrong in not presenting the results and conclusions of my study and observation. And I wish also to enter my

strong protest against the course which is usually followed in regard to cancer, both before and after operation. With a rather extended experience during the last forty years, I have rarely if ever found a patient with cancer who has received adequate and continuous medical care before operation, with a view of discovering and rectifying the cause of the morbid growth. Too often when a cancer is suspected or discovered it is taken as a foregone conclusion that the malady is hopeless, except as the *results of the disease*, that is the new growth, may be removed by the knife, X-ray, radium, caustics, etc. And after a surgical operation, as far as my observation goes, the patients are invariably left entirely to their own resources, with the hope, alas, too often futile, that the tumor will not regrow, but with no attempt to so guide the life that there shall not be the same tendency to a recurrent malignant new-

formation. Against this latter course I also raise my earnest protest.

In presenting matter from literature bearing on the subject under discussion I am well aware that I have hardly more than touched the surface of the deep ocean of recorded observations on cancer, but it was the best that I could do in my busy life. I only hope that others will take up in earnest the subjects here presented, and that this pioneer work may lead to the building of a strong and permanent structure regarding the true basic cause of cancer. Truth never fears proof.

In studying the subject I have been greatly assisted by the masterful work of Roger Williams, so often referred to and quoted from; and I feel that a candid and careful perusal of that book will be a revelation to many. I know that some have endeavored to throw discredit upon some of the deductions which he makes from

various statistics, but in the main they all teach a valuable lesson and are worth serious consideration, as they are taken from reliable sources.

Realizing fully that the problem of the cause and proper treatment of cancer is a most profound one, which master minds have long wrestled with and which cannot be fully settled without much further study, these lectures are presented to the profession at large in the hope that they may be the means of helping some one threatened with or suffering from malignant disease; and also that they may possibly stimulate others to investigate even more diligently along the lines of the medical aspects of cancer.

January, 1915.

531 Madison Avenue.

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CANCER ITS CAUSE AND TREATMENT

LECTURE I

NATURE OF CANCER

It may be safely said that no disease which has afflicted mankind has received as much attention as Cancer, or concerning which there has been as much diligent search to find out its nature and cause. Tuberculosis, which seemed at one time to threaten even the existence of the race, has sunk into relative significance, as we have learned its true nature and conquered some of the causes of its ravages, and reduced its mortality very largely. Syphilis, under various names, forms, and aspects, was formerly much more of a menace than now,

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and in earlier years caused veritable epidemics, but is now well understood and controlled. Leprosy is less of a terror than in earlier times, since it has been definitely shown not to be contagious. Smallpox no longer rages, and yellow fever, and the plague, and hook worm disease have been hunted down by scientific study and the application of proper sanitary and medical measures. And so on in regard to many of the ills which afflict mankind.

But cancer has held its own and has even increased in frequency, with rapid strides and bounds in some localities, until now it looms large as a national¹ or even universal scourge; it has been estimated to cause the death of half a million persons yearly among the civilized people of the

¹ According to the United States Mortality tables for 1912 there were 44,531 deaths from cancer that year, or 77 per 100,000 living, while the rate in 1911 was 74.3, an increase of 2.7. Tuberculosis has shown a steady decline, the death for 1912 being the lowest on record, 149.5 per 100,000, it having fallen from 200 per 100,000 in 1900, or over 25%.

earth, and untold misery and suffering to many times this number. And all this is still going on in spite of the earnest, faithful, and intelligent labors of innumerable research workers, the sacrifice of countless animal lives, and the expenditure of vast sums of money; and the end, as far as relates to its prevention and cure, seems almost as far away as ever, for many surgeons, in past and present times, have acknowledged their inability to cope with the disease.

Much, however, has already been established by scientific research, and still more earnest thought, observation, and endeavor should be given to seeking most diligently for the cause of the disease, in the laboratory as well as in practice; for there must be some cause of cancer, and also some reason for its steady increase.

But it is never to be forgotten that, as Pope says, "the proper study of mankind is man," and clinical observation, with

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laboratory research, on cancer as it appears in the human being, must be the ultimate base upon which all true advance in the knowledge of the nature, treatment, and prevention of the malady can ever rest. To effect this we must study the human being in all relations of life, must know the constitution and class of subjects in which the disease is most apt to manifest itself, understand the chemico-physiologic actions going on in the system, before and during the existence of the disease, and by a process of synthesis and deduction understand what is wrong and endeavor to correct it.

All this is indeed a great undertaking, and can only be accomplished by great effort on the part of many careful and skilled observers. But I want, in these lectures, to give you an outline of my own thought and study for many years, and, if possible, to let you see as I do the lines along which investigation should be directed. It is hoped, therefore, that this and the follow-

ing lectures will throw some light on the connection of cancer with diet and mode of life, and some suggestions as to its prevention and cure.

Parasitism has been excluded: for while at different times many observers have reported various organisms which were thought to be the cause of malignant growths, none of these have been definitely confirmed by others, in spite of earnest endeavor; and all experimental and clinical evidence is against a parasitic etiology of cancer. It is therefore seen how improper it is to speak any longer of "*the germ of cancer*," for, as is now widely acknowledged, there is no such germ, it is an *ignis fatuus* which has been chased in vain.

The *contagiousness* of cancer has also been excluded, certainly in the sense in which this term is applied to other affections. For while in some animals inoculation experiments have resulted in the transmission of certain tumors, little has

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been determined except that such tumor material when transplanted can, in some unknown manner, multiply its cells indefinitely and form a focus of malignant disease, with disastrous effects on adjoining tissue. The same occurs in metastasis in cancer patients. But this does not at all explain the true basic nature of cancer, nor its development in those who have had no connection with other patients so afflicted. On the other hand the instances of suggested or supposed human transmission of cancer from one individual to another are so remarkably few, and so exceedingly doubtful, that a recent author, Janeway, states that "no well-authenticated cases of the transfer of a malignant tumor from one human being to another exist." It has been found impossible to inoculate human cancer into rats, mice, and apes, nor can animal tumors be inoculated into animals of a different species.

Heredity has been advanced as a cause,

but statistics fail to verify this in any degree whatever. While certain instances have been brought forward in which heredity seemed evident, the study of large numbers of those afflicted with cancer, in connection with others free from the disease, has shown almost the same proportion of antecedents with cancer in both classes of persons; although some recent evidence seems to show that there is some tendency in different families for different organs. Experimental studies have, it is true, seemed to demonstrate that tumors occur apparently along hereditary lines in some animals in regard to certain organs; but in these instances it is to be remembered that the animals were kept in captivity, and all fed alike, conditions which have been found to cause the development of malignant disease in wild animals when confined in Zoölogical Gardens.

In former years *malaria* was believed to have an influence in the production of can-

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cer, and some investigators have thought to trace the prevalence of the disease to *tel-luric influences*, showing a preponderance of cases along certain water courses, or in certain streets or houses; but no definite proof of such connection has ever been established, and this theory is dismissed by the best authorities. *Syphilis*, in its latent effects, has also been claimed as an element in the causation of cancer, and undoubtedly the disease may develop, in suitable persons, upon old syphilitic lesions, especially about the mouth, anus, and genital region: but no one well informed in regard to cancer would regard syphilis as the real cause of the disease. All these and other etiological propositions are no longer considered to be tenable, and the very multiplicity of suggested causes shows that we are yet very far from the true etiology of cancer.

Age undoubtedly has a powerful influence in the development of cancer, the vast majority of cases occurring after the age

of forty or fifty. But, again, this does not at all explain the true nature of the disease, for only a certain proportion of elderly people are so afflicted, and malignant tumors have been observed in those of all ages, and even in young children. The degeneration of tissue belonging to advancing years undoubtedly renders it more susceptible to malignant disease, but this does not explain why one person is affected and not another, nor why the tissues in one locality or another take on this morbid action.

More recent scientific study has attempted to show that cancer originates from what are called "*embryonic rests*," or pre-natal, wrongly placed, tissue elements, which at some time or other take on morbid action and develop into what we know as the various forms of cancer. Williams says, "From a biological standpoint tumor formation must be regarded as a phenomenon of the same order as reproduction in general: that is to say, as a spe-

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cial form of overgrowth of the individual.” But here again it is necessary to determine what causes them at certain times and in certain places to thus proliferate and form new tissue, which then becomes malignant and may proceed to destroy all contiguous tissues, and even to cause death.

Traumatism has been claimed by many as the cause which determines the activity of the misplaced cells, and starts them on their disastrous or rampant course: the various percentages of the cases in which it was believed that traumatism started up the malignant process has varied greatly with different observers, even up to 50 per cent. or more. But it is far from proven that this is always the case, nor does local injury in any way explain the persistency with which malignant disease, when once started, pursues its destructive and even fatal career; nor can traumatism account for the great tendency to recurrence constantly observed, either in the former site

or at some distant focus, through the agency of the lymphatic or vascular system. For of multitudinous traumatisms, even in cancer subjects, how few ever develop into malignant disease!

It would seem, therefore, that for the development of the local manifestation of cancer (the tumor or new growth) three elements are requisite, namely: 1. A predisposition or suitable blood condition. 2. A local stimulation or irritation of the part affected, *upon*, 3. The site of an "embryonic rest."

On the basis of the embryonic theory surgeons have of late most earnestly advocated the very early and complete removal of malignant lesions, including those of suspected malignancy, and even also the removal of many innocent lesions which are observed occasionally to lead to cancerous formation; and unless a better plan can be determined this cannot be urged too strongly in proper cases.

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But while early operation has improved surgical statistics, it has not contributed to our real knowledge of the basic cause of cancer, nor has it taught us why those lesions or "embryonic rests" will remain quiescent for years, or prove harmless in some individuals, while in others they become most formidable agents of destruction. For it is now recognized that these wrongly placed tissue elements are very common anatomical or histological accidents, indeed it is claimed that they occur and exist in every individual: and the removal of isolated "embryonic rests" which have developed into cancer, does not by any means prevent the transformation of other similarly misplaced cells into malignant disease, as the frequent recurrence of cancer after operation demonstrates.

The same is true of the many and various forms of treatment other than surgical excision, such as deep acting caustics, and even the X-ray and radium, which like

surgery, only remove the focus of possible systemic infection, and do not affect the basic cause of the complaint: this latter is being shown more and more, by scientific investigation and observation, to be associated with metabolic or chemico-physiological changes in the system, and evidence is accumulating that it is dependent upon them.

All this leads thoughtful persons to inquire if there is not some deeper, fundamental cause lying back of the trouble, which should be reached and rectified by medical skill and acumen, something to do with the life or diet of a person which renders the tissues liable to take on malignant disease. So that a recent surgical writer on cancer states that "all study, whether clinical, pathological, or experimental, points to the fact that there is some underlying, hidden cause which leads to that aberration in the action of tissue cells which we call cancer," . . . a cause

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“residing in only the cells themselves or in some abnormal chemical constitution of the plasma bathing the cells, or in both of these possibilities acting together.”

Occupation has been questioned, but with most unsatisfactory results, for in some statistics which have been gathered cancer has been observed in those following all possible pursuits: and while laborers stood first on the list, clergymen stood fourth, while carters, threshers, and guides, who would be exposed to local injury, were at the bottom of a long list. It has been found, however, to be more frequent in trades or occupations in which the individual is most subject to the habitual abuse of alcoholics, as in bartenders, printers, etc.

We see, then, that thus far no satisfactory cause has been established for the occurrence of cancer, much less for the steady and great increase of the disease of late years. And as far as can be learned,

no measures are recognized, or at least generally adopted, to prevent its occurrence or recurrence; although, as already stated, modern surgery has seemed to improve the statistics in regard to its mortality in certain forms or locations, and the X-ray and radium have certainly also been able to remove, perhaps temporarily, some of the products of the disease.

We come then to the question, what is the real nature of cancer? Alas, all scientific, experimental, and clinical investigations have failed to solve the problem, except that all "evidence points to the conclusion that cancer is to be considered as a pathological disturbance of the normal cell life," from some unknown cause. A curious suggestion has been made by Schmidt, who found that of 241 cases of cancer of the chylopoietic system, 180 had never had any infectious disease of childhood, and 99 went through life without any infection of any kind; the figures point to

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the existence of a cancer diathesis—one which is resistant to germs.

It would carry us too far from the practical side of our subject, even if we were at all able, to present or analyze the vast number of contributions which have been made to the pathological histology of cancer, and the changes which take place in the transformation of normal cells into those of malignant character: the amount of microscopic work which has been done along this line can hardly be imagined, and the literature relating to it is enormous.

Ewing, accepting the definition that the cancer process is “atypical and destructive proliferation of epithelium,” quotes Ribert as saying that “no one has ever seen the beginnings of mammary cancer”: but he does not bring us much nearer to the solution of the cancer problem than we were before. Bainbridge rejects all possibility of a blood condition, and finds the only solution of it in the early removal of every-

thing which is thought to lead to cancer, even the simplest benign new formations, but Ewing states that "in some cases carcinoma has developed after excision of wholly benign fibro-adenoma": and the immense number of cases of recurrent cancer after operations shows that we must look further than surgery if we wish to stay the progress of this formidable disease.

It would be useless to attempt to present the many theories which have been advanced relating to cellular metaplasia, or even to detail all the more or less accepted views in regard to the manner in which normal cells change and degenerate into those of malignant character: but some of the principal facts may be of service in understanding somewhat of the mode of development of malignant tissue from that which has been normal.

The statement of Virchow, "Omnis cellula e cellula," that is, "where a cell

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arises there a cell must have previously existed, just as an animal can spring only from an animal and a plant from a plant," forms the basis of all study on the genesis of cancer and all tumor formation; karyokinesis, or indirect nuclear or cell division, is at the bottom of all growth, both normal and malignant, and the two classes of growth differ only in their methods and activity. In healthy tissues cell proliferation and destruction proceed in an orderly manner, forming homologous structures, as when the hair and nails are constantly produced from newly formed cells at the root, and the result of this new growth is removed mechanically when the hair and nails are cut from time to time, or the hairs fall out. In the case of the skin the epidermal layers are pushed forward and finally exfoliated as useless epithelial débris.

With the cells composing other, or internal structures, however, the process is different. For here each normal cell repro-

duces others of homologous structure, and the different parts of the system are thus kept in active service through anabolism, by means of which new cells are formed with renewed vitality, and the older, or effete cells are removed by catabolism; the elements of which they are composed are thus split up into their component parts, and carried off by the blood or lymph stream, and are then either discharged as effete substances or reutilized in the system, along physiological lines. Wakefield has pretty clearly shown that the developing cancer cell is the product of sub-catabolism, or a sub-oxygenation, induced by hyperacidity or oxidase deficiency in the surrounding medium of the blood plasma.

A great deal of thought, study, and speculation have been given in regard to the behavior of the cells themselves, and strong arguments are adduced for a local cell pathology, that is, regarding the cells as "autonomous beings, possessed of mor-

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phological and physiological independence." But on the other hand we must recognize some restraining influence which continually causes the great mass of cells to reproduce those of homologous structure, in an orderly manner, and only very rarely some of them to break loose and form tumors, which may then become malignant and even destroy life. How this restraining influence is modified or withdrawn is, of course, a part of the problem of cancer. Those who maintain their autogenous character lay great stress upon the polarity of cells, and the relation of the centrosome to the nucleus, indicating a change in the polar axis in cells about to be cancer-genetic, as does Ewing in his recent classical study of pre-cancerous lesions. But whatever changes are observed microscopically we must recognize that the cells themselves must be influenced ultimately by that mysterious force which we call life, which ends with its extinction

from the body as a whole, and which is ultimately related to nerve action. We must also recognize that the cells everywhere depend for their life and activity upon the plasma in which they are bathed and from whence they draw their sustenance; and this plasma is renewed day by day from the food and drink taken.

Students of cancer are more and more recognizing and acknowledging that "all these phenomena, apparently so different, are merely protean manifestations of one common process which underlies and is the cause of them all, to wit, cell growth and proliferation. The particular outcome of the process in any given case is due to the influence of the conditions of nutrition—understanding by this term the whole of the material changes wrought in the organism through its relation with the surrounding world. This being so it is easy to understand how, under favorable conditions, certain cells may take on independ-

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ent action, growing and multiplying without regard to the requirements of adjacent tissues and of the organism as a whole.”

There seems to be some reason to support the view advocated by Williams, that tumor formation and growth are but forms of agamogenesis, or non-sexual reproduction of cells, distinctly related to the decline in growth of the body in general. Hence while the forces of growth, development, and reproduction are in greatest activity the tendency to this disease is relatively small: but when growth declines and nutrition is relatively low the cells undergo gemmation, owing to perverted nutriment, and thus hyperplasia and not inflammation is the starting point of every neoplasm.

Experimental work has time and again demonstrated that cell growth, either good or bad, is modified in no uncertain way by the character of the nutrition furnished. Cancer has repeatedly been observed to disappear spontaneously, as such cases are

on record by careful and competent medical men: in certain of these instances it has occurred in connection with a radical change in the mode of life and diet, but in the majority of instances there is no record of the special cause of its disappearance. The lesson to be learned from this is that there are conditions of the system which are antagonistic to the abnormal proliferation of cell tissue, even when it has begun to take place, as we must believe that there are other conditions of the system which favor such diseased action of aberrant cells.

An interesting confirmation of this is attributed to Ehrlich, but I cannot find the original reference. He "has shown that mice living upon a rice diet cannot be inoculated with cancer, while mice living on a meat diet can be readily inoculated, cancerous tumors developing quickly and continuing to grow until the animal dies. Ehrlich also found that when mice with

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cancerous tumors, the result of inoculation, were placed upon a rice diet, the tumors ceased to grow, and in many cases degenerated and disappeared." Valuable corroboration of this has been given by Sweet, Corson, White, and Saxon. They made a series of experiments in regard to the "influence of certain diets upon the growth of experimental tumors," all with the same results. Of fifty white mice, 25 fed on glutenin and gliadin, and 25 on normal diet, 23 of the 25 on normal diet acquired tumors, against only 4 in the 25 on the glutenin and gliadin. This was repeated on 50 males, with the result of 18 in 25 against 3 in 25: and in a third series, of 50 females, the figures were 15 in 25 against 7 in 25. Thus, they found that 75 per cent. of 75 mice developed experimentally inoculated tumors when under normal diet, while only 19 per cent. of other 75 mice developed such tumors when under a diet of glutenin and gliadin, that is, vegetable proteins;

moreover, the tumors in the latter were in 30 days hardly larger than those in the former in 10 days. Rous has recently shown that large growths of certain transplanted rat and mouse tumors are checked in their development by underfeeding the host on a special diet.

The chemistry of cancer has been studied in most varied directions, and the literature relating thereto is very voluminous and can be hardly more than alluded to here. It is unfortunate, however, that most of the researches have been made in connection with patients who have advanced cancer, and very commonly with the disease affecting internal organs, which of itself interferes very greatly with their function, and so causes many of the perturbations of metabolism observed. What is needed are researches in regard to the metabolism of patients before the development of the disease, or in its earlier stages, before it has exerted its injurious effects

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on the system, in order to learn of the causes which lead up to and induce the wrong action of the cellular elements, whose invasion and malignant action subsequently become so serious.

It is quite impossible in these lectures to enter at all fully upon the various biochemical studies which have been carried on in regard to cancer, but brief mention will be shortly made of some of the salient points. Not only has the structure of carcinomatous tissue been examined chemically, but the blood and urine have been submitted to most painstaking investigation, and metabolism in general has been studied in almost all possible directions, in the search for the cause of cancer; and yet, as Beebe says, "no phase of metabolism has been described in cancer which does not have a counterpart in non-cancerous conditions." But, as previously mentioned, all these observations and studies have been largely made upon advanced cancer

cases, when the system has already felt the unsettling and depressing effect of what is probably an injurious secretion from the deranged and actively proliferating cells of the cancerous mass. In a later lecture we will consider some of these matters in so far as they have relation to the dietetic and medical treatment of cancer.

The essence of our study thus far has been, that in every instance what is called malignant disease is but an aberrant action of originally normal body cells. That, as normal cells find their nutriment in the circulating plasma, so some pathological change in this latter causes them to take on abnormal action, and they no longer develop homologous cells, capable of forming normal tissue, but heterologous elements which have a natural tendency to disintegrate or break down, and exert a destructive influence on adjoining cells of any kind; and in this process they secrete a hormone which is prejudicial to the system

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and tends to destroy life. In later lectures we shall endeavor to understand this more perfectly, and consider some of the elements in life which tend towards the production and arrest of cancer.

LECTURE II

FREQUENCY AND GEOGRAPHICAL DISTRIBUTION OF CANCER

CANCER in man exists all over the globe, but in different degrees of frequency, according to varying conditions of life, as we shall presently see. Malignant growths occur also in animals and fishes, though also with greatly varying frequency under different conditions; but there are few real tumors in reptiles or amphibians. Tumors are also occasionally found in vegetable organisms, presenting increased growth and proliferation of cells, arising from adventitious, or abnormally evolving buds, as also from parasitic and other external irritants. While these vegetable tumors are very interesting and in a measure instruc-

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tive, in regard to the peculiarities of cell growth which they exhibit, they bear, of course, no relation to cancer in the animal kingdom, although some have endeavored to argue otherwise. There is, however, a certain suggestion of analogy to be found in the observation made by one writer, that "the origin of buds, as well as their subsequent development, is chiefly determined by the conditions of nutrition. Wherever there is an excess of nutritive material, capable of being utilized for growth by the cells of the part, there buds may arise"; we shall see later that the same thought applies to cancer in man and animals, when we come to the consideration of the relation of overindulgence along certain lines of eating and drinking to cancer.

Cancer has well been styled a disease of modern civilization, like tuberculosis, although of quite a different nature. Interesting studies have been made in regard to the increased death rate from the former

in England, coincident with a diminished mortality of the latter, in accordance with nutritional changes which have taken place in certain populations: and in the first lecture I mentioned that in the United States the mortality from tuberculosis had fallen 25 per cent. between 1900 and 1912 while, as we shall see later, the mortality from cancer has certainly risen.

Williams, who quotes very largely from the accurate statistics which have long been carefully recorded in England, says that "while tubercle has declined with great rapidity, cancer has increased at a still faster rate, and these inversely related changes are still in active progress. In illustration of these remarks it may be mentioned that during the last half of the nineteenth century, the cancer mortality for England tripled: while, during the same period the tubercle death rate declined to the extent of nearly one-half. Unless some great change in the national

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habits takes place, of which there is at the present no well marked indication, cancer will ere long claim more victims than phthisis, as is already the case in many localities—e. g., Hampstead, Clifton, Bath, etc.” He further says, “I regard this decline in the presence of tuberculous diseases as the direct outcome of the better food and improved hygienic conditions, for which we are indebted to our increased national prosperity: and I shall endeavor to show that conditions of this kind, by their action in another direction, are also mainly responsible for the augmented cancer mortality.” We shall see later that cancer has asserted itself where modern civilization has augmented the opportunities of over-indulgence along many lines of eating and drinking: for while advancing scientific knowledge has undoubtedly diminished mortality in general, and has added to the average length of life, the various factors included in our modern mode of living have

also with certainty increased morbidity along such lines as neurotic and vascular disorders, tumors, etc.

All statistics from various localities show that cancer has certainly increased in frequency very greatly of late years, and though some have attempted to claim that this increase is only apparent, and is due to greater accuracy of diagnosis, and the prolonging of more lives to an age when cancer is more common, there is no doubt in the minds of those who have studied the figures that the increase is certainly very real; and unless there be found some way to check its production, the death rate at the end of the century will be appalling.

It is quite impossible here even to give a comprehensive idea of the immense amount of work which has been given to the study of the statistics of cancer in various parts of the world, as collected in the remarkable works of Roger Williams and Jacob Wolff, but brief mention must be

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made of some of the items observed and recorded in order to properly understand our subject. Williams in particular has analyzed the recorded facts in an interesting and convincing manner, and shown again and again in connection with the figures from different countries, sections, and cities, that the occurrence of cancer bears a striking relation to the condition of the people in reference to their material prosperity; namely, that the well-to-do, who can overindulge in many ways are vastly more subject to cancer than those in the poorer walks of life; also that aborigines in the wilder parts of the world are either almost exempt from cancer, or suffer from it to a very much less degree than civilized foreigners who come to their lands. This is also shown in a very striking manner by Wolff, and I present here a table which he gives in regard to the progress of cancer in a single country, Australia, among the native born and foreigners.

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OF 100,000 LIVING THERE DIE OF CANCER IN
AUSTRALIA

<i>Year</i>	<i>Number of Inhabitants</i>	<i>Native Born</i>	<i>Eng- lish</i>	<i>Other Nationalities</i>
1851	403,889		28	14
1861	1,153,973	5.6	30.5	19
1871	1,168,377	9.7	56.7	25
1881	2,252,167	16.8	72.9	32.6
1891	3,183,237	19.8	119.8	45.9
1901	3,771,715	22.6	203.1	57.3

He remarks, "We see from this comparison in what a great degree the death rate from cancer has increased in foreigners as compared to the native born, in whom the disease has remained about stationary, when the increase in population is considered." Another writer remarks that when native Australians mingle with foreigners as servants or employés, and adopt their diet and customs, cancer occurs more frequently in them. Much the same has been reported in regard to other peoples and nationalities, and later we will consider the influences of urban life on the production of cancer.

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In New Zealand, according to Hislop and Fenwick, where the general death rate is the lowest in the world, cancer is on the increase, as civilization advances. In the great majority of cases the alimentary canal is the seat of invasion, even in women: all the patients studied were hearty eaters, taking also very much strong tea many times daily.

The Polynesians and Melanesians seem to be peculiarly exempt from cancer. Sir William McGregor, although he had operated several times on whites in the Fiji Islands, never remembers operating on a Polynesian or Melanesian, who are practically vegetarians. He never saw a case in British Guinea in 9½ years, and then saw an encephaloid cancer of the tibia in a Papuan, who for 7 or 8 years had lived practically a European life, eating canned Australian meat daily.

In regard to Africa, Williams quotes Dr. Madden of Cairo, who says, "The con-

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sensus of opinion among medical men in Egypt is, that cancer is never found, either in male or female, among the black races of that country. These include the Berberines and the Sudanese, who are all Musulmans, and live almost entirely upon vegetable diet." Of 19,529 deaths among natives of Cairo during 1891, only 19 were due to cancer (females 10, males 9) or 1 in 1028. In England during the same year the proportion of cancer deaths to total deaths was 1 in 29. In the Islands of Lagos, on the West Coast of Africa, Dr. Johnson, in 14 years' practice there saw 5 cases of cancer in natives all of whom lived as Europeans. In southern Africa, "among the Boers and Europeans, who are large flesh eaters, malignant tumors are common: but among the natives, who are mainly vegetarians, these tumors are so rare as to be almost unknown."

Renner reports interestingly in regard to cancer among the descendants of liber-

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ated Africans or Creoles, in Sierra Leone, Africa. During 30 years, from 1870 to 1900, there were but 20 cases recorded as malignant disease among 22,453 admitted to the Colonial Hospital: in the next ten years there were 26 among a total of 10,163, a slow but steady gain in cancer incidence, with the advancing influence of the white man. He says that while the aborigines eat no meat, the "Creoles" eat much meat; the teeth of the latter are beginning to decay, like those of the whites, which is attributed to the sweets introduced by the latter. Every case of cancer recorded has been in a Creole, living like a European, and not a single case among the aborigines.

Much the same freedom from cancer has been noted in regard to negroes when first brought to the United States in slavery, when their food and mode of life was simple: but since emancipation and in proportion as they have mingled with whites and eaten their food, with their own natural

tendency to gluttony and laziness, cancer has increased among them, although their death rate from malignant disease is still much less than that of whites.

In India all writers agree that cancer is rare among the inhabitants of warmer country districts, where they live largely on rice or millet, with a little milk and butter, and vegetables: they eat meat rarely, the immense majority of the people live a rural life, depending upon agriculture for their sustenance.

Investigations of late years, however, might seem to indicate that cancer is more prevalent in India than previously supposed, but its incidence still bears no real relation to that in many other countries, and an analysis of some recent reports explains in an interesting and curious manner the reasons for the diversity of opinion as to the actual frequency of the disease.

Thus, Benratt collected a total of 1700 cases only from 5 years' statistics of 15

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Mission Hospitals and 34 Government Hospitals, representing, of course, many million inhabitants, whereas in New York City, according to the weekly Bulletin of the Board of Health, there were 2193 deaths from cancer in the last six months, a striking illustration of the rarity of cancer in India. Moreover of these 1700 cases, over 1200 were about the mouth, a very large share of these arising from the very common habit of chewing betel, which contains also much calcium, which latter is one of the salts incriminated in the causation of cancer. Sandwith attempts to show that cancer is prevalent in India, but refers to only 2000 cases reported in the hospitals there, in three years, also among many millions of people, and he refers likewise to the betel chewing cancer, and the "kangri burn" on the abdomen of men, from the charcoal furnace worn for warmth: these peculiar local disorders viti-

ate any deductions which could be drawn from such statistics.

In China, according to a recent writer, "cancer is comparatively uncommon in those parts where the bulk of the people live on an almost exclusively vegetarian diet, being too poor to purchase any of the various flesh foods, which are there used for culinary purposes. But in places where cancer is said to be more prevalent, the reporter adds, "All Chinamen there eat fish and pork at morning and evening meals: fowls and ducks are always on the table of all but the most humble of the coolie class."

In regard to the occurrence of cancer in the Far East, however, some of the modern investigators, such as Bashford, have endeavored to overturn the generally accepted view as to its infrequency, but I do not feel that the evidence presented can at all weigh against the unprejudiced opinion

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of most capable medical men who have long lived and practiced in those regions, some of whom as medical missionaries have had most intimate contact and acquaintance with the natives. Only very recently a medical missionary, who has long been connected with the medical college and hospital in Beirut, Syria, told me that cancer was practically unknown among the thousands of patients who flock there from all over the Near East, he adding that they were all largely vegetarians.

During a rather extensive trip through the Far East I was unable to see or even hear of any cancer, although I met a large number of medical men, and made diligent inquiry regarding the same. As I wished to verify my views in regard to the rarity of the occurrence of cancer among those who lived on rice or other vegetarian diet, I visited very many civil, military, and mission hospitals, with a total of many thousands of patients, and ministering to

many millions of population; in Japan, Korea, China, the Philippines, India, Siam, and Egypt, I met the same response, that cancer was rarely seen among those vegetarian natives.

Brazil is credited with having the lowest cancer record of any portion of the western hemisphere, especially among the natives in the Equatorial regions, while in the Argentine Republic, where meat is known to be largely consumed, cancer is fairly common. From many parts of the world there come reports of the relative infrequency or even absence of cancer among simple living natives, one writer in regard to the West Indies stating "Even those cases which I have witnessed in this class of people have been among the better orders of them, whose habits of living assimilated to those of Europeans."

England and Wales present the most satisfactory field for the study of the progress of cancer, as the national vital statis-

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tics have been well kept since 1840; even at that time under the able direction of William Farr they had already acquired a well-deserved reputation for reliability, as Williams remarks, from whom I shall freely quote.

In that year, 1840, there died of malignant disease in England and Wales 1 in 5,646 of the total population, 1 in 129 of the total mortality, or 117 per million living. In 1905, the deaths, due to this cause were 1 in 1,131 of the total population, 1 in 17 of the total mortality, or 885 per million living: thus, while the population had only a little more than doubled, the cancer death rate per million living had increased five fold. Dr. Williams answers by figures and tables the several objections which have been raised in regard to the actual increased mortality from cancer, as it has been repeatedly claimed that the increase is only apparent and not real; thus it has been asserted that it is due—1. To mere

increase of population: 2. To the average age of the population having advanced: and 3. To improved diagnosis and more careful death certification. Time does not admit a full presentation of his statistical refutation of these claims, to which he devotes some pages very convincingly, but it can be safely accepted that for some as yet unknown reason, cancer has made strides in England which are truly alarming.

Williams has also made some most interesting studies in regard to the increase of cancer in connection with changed conditions of life, and from his analysis of statistics, he very clearly shows that the spread of the disease has closely followed urbanization, and the rapid increase in material prosperity of recent years: in England where 80 per cent. of the population are now town dwellers, this tendency to collect in cities and towns has gone farther than in any other community. He recognizes that any far-reaching, environmental

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change of some duration is probably potent in disturbing the stability of the constituents of living bodies, and the sudden change from poverty to riches and plenty is conducive to the development of cancer: allusion has already been made to the inverse relation of deaths from cancer and tuberculosis, the latter diminishing with improved material conditions, while the former increases as wealth and indolence increase.

He shows this by statistics from various localities, and by data from towns in different countries he makes it pretty clear that "Cancer mortality is lowest where the conditions of life are hardest, the surroundings the most squalid, the density of population greatest, where the tubercle mortality is highest, the general and infantile mortality greatest, and where sanitation is least perfect—in short, among the poor of the industrial class in our great towns: whereas among the wealthy and well-to-do, where the standard of health is at its best and life

is easiest, and where all the conditions of life are just the reverse of the foregoing, there the cancer mortality is highest."

While this is a pretty strong statement and many exceptions could undoubtedly be found, careful investigation will show it to be true in the main; for it must be remembered that even among the poorer classes gluttony, especially in regard to proteids, is not at all uncommon, and indolence, with impeded metabolism, is not at all unusual. Dr. Latham found that the mortality from cancer in England, from 1881-1890, was more than twice as great among well-to-do men having no specific occupation, as among occupied males in general, the respective mortality ratios being 96 for the former and only 44 for the latter. Sir William Banks confirms the steady increase in cancer very strongly, which he attributes to richer and more abundant food, of which males eat more than females, and consequently cancer is increasing proportion-

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ately more among men, as all statistics show.

Switzerland is reported to have the highest death rate from cancer of any country, it having augmented from 114 per 100,000 living in 1889, to 132 in 1898. There again the cancer mortality varies greatly in the different sections or cantons: thus, in wealthy Lucerne it is 204 per 100,000 living, and only 36 in poverty stricken Valais. In the city of Geneva it is 177 per 100,000 living.

Denmark, next to Switzerland, is reputed to have the highest cancer death rate of any country in Europe, viz.: 130 per 100,000 living in 1900. But here the statistics are only from the towns, which comprise but a quarter of the whole population: the per capita wealth is said to be higher there than any other country in Europe except France.

France shows a high cancer mortality, with a constantly increasing death rate;

and, next to England, France is the richest country in Europe, and wealth is much more widely diffused: the French workers own nearly 8 times, per capita, more than those in England. In Paris the cancer death rate has increased as follows, for each 100,000 living, in 1865, 84; in 1870, 91; in 1880, 94; in 1890, 108; in 1900, 120.

Italy, a comparatively poor country, shows a low cancer mortality, but even here it is increasing from 20 per 100,000 living in 1880, to 52 in 1899, and 58 in 1905. The consumption of meat is there the smallest in any European nation, namely 23 pounds per capita in 1895. In the chief towns the rate of death from cancer is high: thus for each 100,000 living, in Florence 137, Ravenna 120, Venice 103, Milan 101, and Rome 77.

Time does not permit a wider survey of the field of distribution of cancer, as presented so remarkably from official statistics by Williams, and Wolff; but in con-

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nection with the high percentages of deaths above quoted among the richer classes it may be interesting to mention some of the lowest records. Thus, in the poor country of Kerry, Ireland, it was 27 per 100,000 living, in the province of Dalmatia 19, in the Shetland Islands 16, in Servia 8 (from 1895 to 1904), and in Ceylon in 1903 the mortality from cancer was about 6 for each 100,000 living.

The United States, unfortunately, has not kept the vital statistics of the country in years past with anything like the fullness and accuracy which has obtained in England, nor even at the present time is it possible to learn definitely the frequency and increase of cancer in every locality. But all the statistics which have been gathered show unequivocally that the disease has steadily increased in a manner which is alarming. Analyzing the recorded deaths from cancer in thirty-one cities, and the percentage of increase in four years,

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one writer estimates that, if the same increase is continued, by the end of the century there will be a death rate, approximately, of 1000 in every 100,000 inhabitants, or one in every hundred.

In a recent Bulletin of the Board of Health of New York City the following statements are made in regard to the mortality from cancer in 1913: "The statistics of our seven largest cities recently tabulated, show that the cancer death rate was the highest on record. For New York City the rate was 82 per 100,000 of the population, against an average of 79, for the last five years: for Boston 118 against an average of 110: for Pittsburg 79, against an average of 70: for Baltimore 105, against an average of 94: for Chicago 86, against an average of 81: for Philadelphia 95, against an average of 88: for St. Louis 95, against an average of 85." This average increase of almost 8 per cent. of deaths from cancer in the combined population of

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These seven cities, during the last five years, have certainly an alarming fact, and cannot be explained on the ground of greater accuracy of diagnosis: for it is not to be presumed that there has been such great improvement along diagnostic lines during the longer war 1913.

It is difficult to state the exact prevalence of cancer in the entire United States. The "registration areas" include only about one-third of the total population: but it can be learned, however, from the annual statistics published since 1900. According to the Mortality Statistics of the United States, the deaths from cancer and other malignant tumors per 100,000 population were 63, in 1900, 63, in 1904, 70, in 1908, 75, in 1912 there were 80 deaths from cancer, or 77 per cent. for the whole country, an increase in the death rate from this disease of almost 25 per cent. since 1900, and, as before stated the

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tuberculosis mortality had fallen a little over 25 per cent. in the same period.

As in other countries, which might also be expected from the statements already made, the disease varies in frequency in different localities and communities. Thus, cancer is stated to be much more prevalent in the northern than in the southern states, and as already stated, the negroes are much less subject to the disease than whites, especially when they are living their own natural home life; but when they come to the cities, as waiters, etc., in hotels, their cancer death rate increases. But even in New York City in 1912 the deaths from cancer in negroes was 1 in 32.2 total deaths, against 1 in 17.7 in whites; the mass of negroes here, of course, live plainly and work hard. The North American Indians also are believed to be almost exempt from cancer in their primitive savage condition, but as they have come under the

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influence of civilization they are more affected. It has also been noted by several observers that immigrants and their descendants present a very much higher mortality from malignant diseases than prevails in their native countries; from these and other considerations Williams suggests that abrupt change of environment may also be a factor in the causation of this disease.

We have thus seen while cancer is very widely distributed over the globe it is present in varying degrees of severity in different localities, and careful analysis shows that the disease affects different classes of persons with unlike severity. All these statistical studies and observations serve to confirm the statement made earlier that cancer is a disease of so-called civilization, and that it has increased in proportion as human beings have come under the influence of wealth, and consequent luxury and overindulgence, with bodily inactivity; all

these elements lead to a disturbed metabolism, which as we shall see later, is, at least, a contributing cause to the deviation from normal of some of the cellular elements of the body. It also appears that some of these metabolic shortcomings have to do with a disturbed nitrogenous balance, which is due to the constantly increased consumption of meat. In 1909 the meat consumption in the United States had reached the high figure of 172 pounds per capita, as I learned recently from Washington, a far greater amount than in England, 130 pounds, as already stated; and with this steady increase in the use of nitrogenous food cancer has also increased by leaps and bounds in both countries.

LECTURE III

METABOLISM OF CANCER

IN the first lecture we saw that cancer was an alteration of the normal cells of the body, whereby they take on a malignant action and continue to do so, destroying contiguous tissues and leading to a lowered vitality, with an apparent poisoning of the system, which finally causes death. As the cells of various organs furnish different secretions, which in health contribute to proper metabolism, resulting in growth or maintenance of the tissues, so these disordered cells are believed to secrete a toxic substance, or malignant hormone, which has a prejudicial action on the body, and hæmolytic action on the blood, as has been brought out pretty clearly by Troisier and others.

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We saw that as yet the definite cause had not been determined, why at some period certain cells take on the action which we call cancer, nor why they persist in their destructive course. Long continued and abundant laboratory and clinical research have about decided certain questions negatively in regard to its etiology, so that in a measure the field is cleared for the study of some of the possible basic causes of the disease in question. Thus, all are pretty well agreed that cancer is *not* contagious or infectious, that it is *not* caused by a micro-organism or parasite, that it is *not* wholly due to local injury, that it does *not* appertain to any particular occupation, that it is *not* hereditary to any great degree, that it does *not* especially belong to or affect any particular sex, race or class of persons, *nor* is it confined to any location or section of the earth, and that it is *not* wholly a disease of older age.

We saw further that there appeared to

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be good evidence that certain misplaced "embryonal rests" were the original starting points of diseased cell action, but as these are now known to exist in every one from birth, this offers no real explanation of the occurrence of the disease at different times in life. It is, of course, quite possible that local injury of one kind or another may be the exciting cause which determines that a cell or group of cells shall revert to its original reproductive activity, as Williams contends that the process is one of agamogenesis, dependent upon excessive and faulty nutrition. The question as to the relation of uricacidæmia, or lithæmia, to cancer has never been fully studied, and it is worth considering whether, as in gout and rheumatism, to which cancer is often associated and perhaps closely allied, the exciting cause may not be the lodgment somewhere of uratic deposit, which is further excited and fed

by effete or imperfectly oxidized nitrogenous elements; for later we shall see that perverted metabolism, largely of proteid elements, is closely associated with cancer.

We noted also that some attributed cancer to independent cell action, relating to the polarity of cells, etc.; but it is inconceivable that a cell or cells can idiopathically start out on a rampant course and pursue it with increasing severity, even until death results, without, at least, some definite pre-disposing cause, even though diligent and earnest work has not as yet determined just what that cause may be. The error has been, we believe, in searching too exclusively by the microscope and by certain laboratory methods, and not sufficiently along clinical and bio-chemical lines. For it must be recognized that all the cells of the body are continually bathed in the vitalizing fluid of the blood, whence they derive their nutriment, and into

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which, with the lymphatics, they return the products of their vital action, by anabolism and catabolism.

By exclusion, therefore, we are reduced to seek the etiology of cancer along other lines, and about all that is left is metabolism, as influenced by advancing, so-called civilization, which relates very largely to diet and mode of life. This we will take up later, but will first examine some of the scientific findings in regard to the blood in cancer, and data relating to the various secretions and excretions of the body bearing upon metabolism in this disease.

That the blood shows great changes in advanced cancer is recognized by all, as is clinically manifested by the intense cachexia and anæmia commonly present and always strongly marked toward the end, of which the cytology has been very fully studied and presented by Türk. When then examined there is found to be a marked reduction of red cells, low hæmo-

globin index, and distinct leucocytosis, with greatly diminished alkalescence.

The reported changes in the blood have also varied with the location of the malignant disease, according as it may interfere mechanically or otherwise with the function of certain organs, which fact naturally obscures the question of the true relationship of the blood to cancer. Thus, it is stated that in cancer of the liver and pancreas there is always leucocytosis and glycogen, and that "cancer appears to interfere greatly with the function of the liver as a destroyer of intestinal toxins, they pass into the general circulation, probably cause the glycogen reaction, and at least part of the leucocytosis, and very often give rise to fever." There are also other microscopical alterations in the blood in late cancer. Thus, degenerative change in the leucocytes are common, with derangement in the normal proportion of their different forms, as also changes in the ery-

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throcytes, with nucleated red cells and megalocytes in severest cases.

Price Jones in a study of the blood in 30 cases of cancer (9 of the breast) found the red blood cells diminished on an average of 6 per cent., the white blood cells increased 38 per cent., lymphocytes increased by 10 per cent., large mononuclear cells increased 164 per cent. and polynuclears 42 per cent. Burnham states that in the severe grades of anæmia with malignant disease, poikilocytosis is marked, and nucleated cells of both normoblastic and megaloblastic type may be present. The red corpuscles may be reduced to 2,500,000, and exceptionally to 1,000,000. Cohnreich in a very technical study of blood from cancer subjects, observed very great increase in the resisting power of the red blood cells to osmotic tension, that is, in regard to their hæmoglobin, which he believed to be of diagnostic value in doubtful cases.

Unfortunately, there have been relatively

few studies of the plasma of the blood in this or other diseases; and yet the condition of this fluid must be of the utmost importance, as from it are derived the nutrient principles not only of the solid constituents of the blood, but also those of the entire system, about 8 per cent. of it being serum albumen and serum globulin. It also holds in solution the phosphates, carbonates, sulphates, and chlorides, the latter often varying greatly, and being chiefly responsible for the isotonic relation of cells and serum. In cancerous cachexia a diminution of carbonic acid, a constantly diminished alkalinity, and an increase of acid principles of the blood have been fully demonstrated, pointing in all probability to the existence of an acid intoxication. The formation of the corpuscular elements of the blood must be greatly interfered with when metastases occur in the blood making organs, the lymphatic tissue, bone, marrow, and spleen, which probably occur

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more frequently than is generally recognized. It seems that the toxic secretion from a cancerous mass has a distinct action upon the blood, for after complete removal there is often observed an increase of hæmoglobin, as I have witnessed, and a high leucocytosis has disappeared after the removal of schirrus of the breast, only to return again with the recurrence of the tumor. Abderhalden states that in from two to three weeks after the operative removal of cancer, certain defensive ferments can no longer be found in the serum.

Many laboratory studies have been made upon the chemistry of cancer tissue, seeking to determine the nature of the toxin produced, and its experimental effect on animals, but thus far no great results have been obtained. It has been observed, however, by Gruner that when cancer juice is injected intra-venously a marked lymphocytosis arises, which is followed by the appearance of large mast cell myelocytes in

the blood. This cancer juice is supposed to be autotoxic in cancer patients, and to comprise toxic albuminoids, which being in quantities too great to be quickly neutralized poison the system, especially the blood and the hæmatopoietic organs.

In regard to the real bio-chemistry of cancer, we are still greatly in the dark. Vast numbers of studies and researches have been made to determine the real character and nature of the bio-chemical changes which occur in cancerous tissue, and the mere recounting of the reported findings and theories elaborated from them would occupy far more time than can be profitably given in these lectures. Some have claimed very positive findings which account in a measure, at least, for the pathological conditions, while others, as Beebe, state that "the chemical study of tumors is in its infancy. We have scarcely proceeded far enough to know where the medical problems are, nor have methods

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now available been perfected to such an extent as to enable a decisive experiment to be made." "No phase of metabolism," says he, "has been described in cancer which does not have a counterpart in non-cancerous conditions. This applies to such questions as the nutritive relations between the cancer cells and the normal body tissue, to the nitrogenous balance, retention, elimination of sodium chloride, excretion of acetone, the relation of ammonia excretion, and a possible acidosis." He adds, however, "Diet doubtless forms an important part in the growth of cancer, possibly even in the origin of the disease." It is encouraging, therefore, to find that this able and careful laboratory investigator recognizes, in a measure, the basic cause of diet, toward which all evidence points so strongly, although the definite connection may not yet have been established by laboratory methods.

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In all our study in regard to the relation of diet to cancer it must be remembered that there are divers elements and agencies which combine to produce the many and various disordered conditions of the body, to which we give the names of different diseases, and that cancer is no exception to this general rule. For instance, in old-fashioned gout the patient may have consumed an excess of Port and Madeira wine for years before the system finally rebelled and acute gout resulted; and among the causes for the systemic reaction we know that frequently it is great mental strain or shock which has so disturbed metabolism that the wine was no longer tolerated. Much the same is true in regard to cancer and nitrogenous diet. And we will see later that mental disturbance and nerve strain or shock often seem to be causative elements; also that constipation, or intestinal stasis, is so common in cancer sub-

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jects that it must be looked upon as one of the contributing causes among others, to be mentioned later.

Although it is quite possible that many of the reported bio-chemical changes found in primary cancerous tissue and metastases may not be of etiological importance, it may be interesting to briefly refer to some of them as indicating the vital alteration in tissues connected with what we recognize as malignancy; even as in acute and chronic gout the affected tissues exhibit abnormal conditions in regard to uratic deposit.

Many writers, some of them dating back many years, agree that albuminous constituents predominate in cancer tissue, and, as in actively growing structures in general, sugar forming substances abound. Wolter states that cancer of the breast contains 20 per cent. more nucleo-proteids than the normal breast. Casein is also present in breast cancers, and the abun-

dance of fatty matters, contained in the cells of such neoplasms, is well known. In regard to the proteids, Wolff, after many studies, concludes that their character is identical with that of normal tissues, and it is only the quantitative distribution of these that differentiates the tumor from the physiological tissue. Wells agrees with others that there is no very distinctive character in the bio-chemistry of malignant tumors, but by reason of their excessive chemical component, as compared with benign tumors, they naturally show a high content of nuclear proteins; they, therefore, contain a high proportion of phosphorus and iron.

Interesting observations have also been made on other characteristics of cancerous tissues, such as the great abundance of enzymes of great variety which are actively autolytic, also in regard to certain relations of cholesterin, in regard to which Ewing has recently said, "There appears

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to be something in the chemical or mechanical nature of the irritation of cholesterol which is peculiarly effective in producing atypical proliferation of epithelium''; this has been found to be no less than 65 per cent. greater in quantity in fatty deposits, as in the mesentery, in subjects of cancer than in healthy persons, etc., etc. It would weary you to no purpose to attempt to refer further to the bewildering mass of research studies in connection with the biochemistry of cancer which are found in special literature: much of it is fragmentary and some of it contradictory, but all has its value as contributory to our knowledge of the actual conditions developed in connection with cancer growth; but up to the present time it cannot be claimed that any very practical results have been thus attained which will aid us in treating the disease.

As all cell life and proliferation of tissue depends on the activity of the cell nu-

clei, much attention has been paid to the changes found in them and the behavior of the centrosomes and chromosomes, all of which is too technical for us to consider here: suffice to say, however, that several observers have demonstrated heterotypic mitosis in malignant tumors, and that histologic examination confirms what other judgment has indicated, namely, that the cancer cell differs from a normal tissue cell mainly in its aberrant action under some stimulus, probably derived from the animal fluids by which it is surrounded. Thus we come back to our original proposition, for these fluids are, of course, but a reflection of the nutrition of the body or diet, as modified by the action of the various organs, including the internal secretions; all this is influenced again by the action of the nervous system.

It is difficult to produce definite proof in regard to the influence of nervous and mental strain and shock in the production

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of cancer, but careful observers have long claimed that there is such an influence, and from what I have seen I am firmly convinced that in some way these conditions often do so disturb the metabolism, or otherwise operate, in such a manner that cancer results. The influence of the mind upon the body is unquestionable, as has been so fully illustrated by Tuke, and from what I have observed I cannot doubt but that the mental depression common in those with the beginning of a process which they fear might result in active cancer, has much to do with accelerating its growth; whereas, on the other hand, the hopefulness which can arise with the attempt to change the diseased process by diet and proper medication, has much to do with the favorable results which may follow in suitable cases. In the same way the constant fear of recurrence after operative removal can have its share in inducing and perpetuating the metabolic

error which excites the tissues to renewed cancerous action. I know that some of you will think that this is fanciful theorizing, but many a scientific fact, in many branches of science, has been worked out from a theory which at first has seemed fanciful.

We will now consider some of the data which have been recorded in regard to the relation of the secretions and excretions of the body to cancer, including the internal secretions.

Much labor has been expended, by very many observers, upon the analysis of the *urine* in connection with cancer, as that might be expected to reveal the metabolic changes connected with this disease. While many departures from the normal have been reported, and while under complete volumetric analysis the urine of a subject of cancer is rarely if ever that of health, it cannot be said that any definite and specific changes have been established

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which may not be found in those without cancer; although there have been several who have so claimed even diagnostic signs from the urine. But minute, volumetric analysis is often of great service in guiding the nutrition and medication of these patients, and gross errors are continually met with, which have the greatest bearing on the case in hand, as indicating very great metabolic disturbance: and constantly the urinary excretion will be found to be extremely deficient, both as to its quantity and its total solid elimination. In one very interesting case of cancer of the breast, in a stout, flabby lady, near 55, in private practice, the total daily quantity of the urine, measured for weeks, is always very far below the normal amount; and in spite of active medication it seems almost impossible to raise the total daily solids excreted in the urine, to more than one-half of that called for by the weight of the patient. We may now briefly consider

some salient points reported in connection with the urine in cancer.

As remarked in regard to other elements in the study of the disease, it would be very desirable to have a knowledge of the urine in pre-cancerous stages of health, or ill-health, and also in very early cancer, likewise after surgical operations, that we might better understand the metabolic changes which lead up to malignant disease; but unfortunately these are exceedingly few and unsatisfactory, and almost all the studies have been made in advanced cancer, and often when the disease has affected vital organs, or when by its own poison it has disturbed the workings of the economy.

Many observers agree that there is a disturbance of proteid metabolism in cancer, and dependent upon this many deviations from normal are found in the urine, some of which in turn are related to the inanition which occurs. The *urea* is almost

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invariably diminished, often very greatly, as I have verified time and again in many cases.

A number of studies have been made upon the nitrogen partition in cancer by Einhorn, Kahn, and Rosenblum, also by De Bloeme, Swart, and Terwen, and others, showing an increase in colloid nitrogen, to more than double the normal amount, increased elimination of xanthin, oxyproteic acid, and urinary ammonia, together with many other changes which show that disintegration of the protein elements is very imperfect and often excessive. An interesting statement is made by Blumenthal that the oxyproteic acids are increased even in very early cancer, and independently of the size of the tumor and degree of cachexia, seemingly showing them to have some specificity for cancer, because they have not been found in other forms of malignancy. He also states that urobilin is increased in a large proportion

of cases of cancer, especially when cachexia is setting in, and is a grave symptom.

Reid, who has confirmed many of these matters reported by others, says: "I have found an increase of amino-acid nitrogen in practically every case of cancer I have examined"; . . . "Hence we can only infer that in cancer, the liver, while not involved in the disease, is still unable, for some reason, to perform its functions in synthetizing urea. The organ is functionally injured, no lesions having been found to explain its insufficiency"; or possibly . . . "cancerous subjects form proteids which the liver is unable to deal with, so that they are excreted unchanged, or nearly so." Degrez has made confirmatory studies along these lines, and found the nitrogen disintegration very imperfect, with increase of the ammonia fraction of nitrogen, and increased elimination of xanthin bases. He states that "the toxicity

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of the urine is increased apparently as the result of the presence of substances which have not been fully oxidized."

Notable changes have also been recorded concerning the *sulphur* elements in the urine, with a great increase in neutral (un-oxidized) sulphur and a considerable excess of sulphy-cyanic acid, together with an increase in sulphates and indican showing the results of intestinal fermentation of protein elements, which also I have constantly observed.

The *chlorides*, on the other hand, are, as a rule, diminished in cancer, especially in its late stages, when there is inanition or kidney insufficiency; and probably any change in them has only a relation to the nutrition of the patients, for the chlorides come from the food and are commonly an index of the amount of nutriment absorbed. Robin finds some relation between the excretion of chlorine and nitrogen, according

to the stage or degree in which the system is affected by cancer.

The *phosphates* are known to be increased in the urine of cancer subjects, although irregularly and in an inverse ratio to the chlorides; as inanition increases there is greater autolysis of cellular structure, and the nuclei yield an excess of phosphates, which are excreted in the urine. A more or less general demineralization of the system through the urine has been observed by several, and has been recognized as a significant matter, and is of special importance when we consider what an important part minerals take in the nourishment of cell life.

While the changes which have been observed in the urine in connection with cancer are not wholly pathognomonic, but occur in connection with other diseased states of the system, so that none of them can be accepted as diagnostic of malignant

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disease, they all have a certain significance as indicating the metabolic changes which accompany and, as we believe, have much to do with the etiology of cancer; and, as stated before, a careful, systematic, and frequently repeated volumetrical analysis of the urine certainly assists greatly in the proper management of these cases, that is when the departures from normal are carefully studied and correctly interpreted.

The *saliva*, and its action, constitutes a very important part in the process of digestion, and consequently of metabolism and the genesis of cancer; far too little attention, however, has been paid to it practically, in ordinary life or disease, although there have been many laboratory studies and writings on the physiological action of this secretion; but I have not been able to find in literature any investigations relating to its condition in cancer. And yet the experience and writings of Mr. Fletcher and others have demon-

strated wonderful results from perfect mastication and thorough insalivation, and a careful consideration of digestion must convince every one of the importance of this secretion in connection with nutrition, both in health and disease.

Our time does not permit of more than a brief allusion to the subject, but in cancer patients I have so constantly found the salivary secretion acid, and often strongly so, instead of the normal alkaline or neutral, that I cannot but believe that this condition has some bearing upon the subject which we are studying; the saliva also is apt to be acid in diabetes, which is closely allied to cancer. It is to be remembered that the saliva, which amounts in health to between one and two quarts daily, varying somewhat with the food, is not wholly for the purpose of lubricating the mouth and facilitating deglutition, but its enzymes, ptyalin and maltase, effect radical and important changes in the starchy matters

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consumed. It is also to be remembered that the latter cannot be acted upon by the stomach secretions, but must be passed on to the influence of the pancreatic fluid in the small intestine, in case the action of the saliva has not been effective; hence there follows delayed and imperfect digestion, faulty metabolism, deranged nutrition, and possibly tumor growth. The importance, therefore, of very slow eating, thorough mastication, and perfect insalivation cannot be too strongly insisted on, both as an element of importance in the prevention of cancer, and also as a curative measure in patients in whom the diseased process has already manifested itself.

The so-called *internal secretions* have also been the subject of much research and speculation of late years, in regard to their influence on metabolism and the life processes of the economy, and many studies have been made concerning their connection with cancer, which cannot be long

dwelt upon now; but there seems to be little doubt but that the secretions of the ductless glands in common have much to do with regulating the metabolism of the cells. We know, for instance, that disease of the pituitary body produces bone disorder, resulting in gigantism, that thyroid derangement results in myxœdema, and that disease of the supra-renal capsules gives rise to Addison's disease, or bronzed skin; and it is not at all impossible that the derangement of secretion of one or more of these and other organs may be an element in the disordered action of certain epithelial cells, resulting in cancer. Harrower calls attention to the fact that cancer is essentially a disease of that period of life when certain of the ductless glands lose their normal function, this loss entailing related changes in the whole chain of interrelated functions of the ductless glands.

The *supra-renal* glands by their secre-

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tion have, as we know, great vaso-constrictor influence, and their complete removal is followed by death; it is more than possible that some failure in this secretion allows the exuberant blood to supply cancerous growths. Sajous says: "Certain growths, particularly the more malignant forms of sarcoma and carcinoma, seem closely connected with adrenal insufficiency and its normal consequences," and there have been some clinical and research data confirming such a conclusion. Sajous further says, "The adrenals, as supporters of the thyroid apparatus in the defensive process, and in sustaining oxidation, metabolism, and nutrition, seem to offer a new clew to the pathogenesis and treatment of cancer that is worthy of further inquiry."

The *pancreas* has been thought to have some influence in a perverted metabolism leading to cancer, Kahle stating that there is a retention of silica in that organ in cancer patients, to even double the normal

amount. A treatment of cancer introduced by Beard, also strongly presented by Saleeby, by trypsin and amylopsin, the enzymes of the pancreatic fluid, excited some attention a while ago; but unfortunately no satisfactory results have thus far been obtained from this line of medication, as was fully demonstrated by Bainbridge at the New York Skin and Cancer Hospital, in one hundred cases.

The *pituitary gland* by its secretion has undoubtedly some coördinating power, with the adrenals and thyroid, over the processes of metabolism, and a number of observers have regarded it as of importance in connection with the genesis of cancer. Little has recently reported some cases of cancer in which *pituitary extract* with that of the pancreas has produced remarkable results.

The *thyroid* is now recognized as playing an important part in assisting metabolism, and, like the adrenals and pituitary,

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its complete removal, with the parathyroids, in animals is followed by death. While the study of the hormones is still in its infancy, there seems to be no question but that the endocrinous glands act conjointly, the one influencing the other, and that together they exert a very great influence in the life processes of the body and on the behavior of its component cells. The thyroid has been shown to be one of the main factors in the management of calcium within the body, which is believed to be an element in cancer, and enhances the catabolism of toxic wastes, which are etiological elements in this disease. Many have reported favorably on the effect of thyroid feeding in cancer, and after an experience with it in many cases I am convinced that it has been one of the means which contributed to the good results obtained. On the other hand it has been claimed by Stuart-Low that the surgical removal of the thyroid, or part of it, or ligation of the

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thyroid blood vessels has arrested cancer, in several cases.

Thymus gland feeding has also been reported on favorably by a number of observers (Rohdenburg, Bullock, and Johnson, also Gwyer), they reporting relief of pain and improved general conditions, notably gain in weight and increased hæmoglobin in all but one of sixteen cases, though some of them died. On the other hand Ross reports most unfavorably on the administration of thymus, which he gave to some inoperable and hopeless cancer cases, in which he said that in two or three weeks the tumors had quadrupled in size and the condition of the patients was very much worse; the same occurred also when some cancer patients were given calcium salts freely. Ross makes some interesting suggestions in regard to the thymus, and its relation to calcium and magnesium; these latter are freely utilized in the growth of bone up to the age of twenty-

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five years, by which time the thymus gland has quite disappeared; but after this time these salts tend to have pathological relations in various tissues, and also cancer becomes frequent.

The internal secretions of the *testicles* and *ovaries* are also thought to have some share in metabolic processes, and observations have been made in regard to their influence in cancer. Thus Cahen reported that Beatson's operation of castration for cancer, done first in 1896, had been repeated by many, so that Lott had reported 99 cases including his own. Of these in 23.2 per cent. the operation caused a distinct improvement in the cancer. In 15 cases the improvement persisted for a year, in 4 cases for 4½ years and in one case for over 5 years. Cahen operated on seven women with remarkable results; in two cases life was prolonged 4 and 6 years respectively. Others, however, have shown by statistics, that damage to the ovaries by

disease, or their removal by operation, greatly increases the proclivity to cancer. Several writers have connected cancer with the waning of the sexual powers, and the suggestion is made by Sherrington and Copeman that in the period which antedates the cancer age, the reproductive glands, by means of internal secretions, are able to inhibit the growth of cancer.

In looking back over what has been observed in regard to the secretions, including those of ductless glands, we see that very strong evidence has accumulated to show that they have a very intimate connection with the development of cancer, as was to be expected, since they are very important factors in connection with metabolism.

We have also seen that while there has not been demonstrated any very definite and specific change in the bio-chemistry of tumors, and no specific enzymes or poison secreted by cancer cells which can com-

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municate the disease, there is evidence that the disordered cells secrete a something which deranges the blood and ultimately tends to end life; for the blood in advancing cancer undergoes very radical, degenerative changes, some of which improve decidedly when a cancerous mass is removed surgically, but return with the regrowth of the tumor.

We have also seen that the urine manifests alterations which show a disturbed metabolism, and that the saliva has an abnormal acidity leading to disturbed amylaceous digestion.

We have further seen that there is evidence that the internal secretions of many organs, probably, through their influence on metabolism, are factors of importance in connection with the genesis and cure of cancer. Little well says, "Cancer is a disease of disordered nutrition, as a result of which cells revert to a primitive stage, which permits reproduction. The disor-

dered nutrition is due to relative hypofunction of the ductless glands." In later lectures we shall consider the basic causes of this deranged nutrition, which, as has been already intimated, has much to do with diet and the various elements of life which tend to induce functional and other derangements of the system, many of which are included in and influenced by what we term the advance of civilization.

LECTURE IV

RELATION OF DIET TO CANCER

IN our earlier lectures we saw that cancer was undoubtedly a diseased action of originally normal tissue cells, due largely to perverted metabolism, the special features of which were brought out last week. In the second lecture we studied the frequency and geographical distribution of cancer, which was found to be very different for various peoples in diverse sections of the earth, and which we saw was proportioned in a great measure according to their diet and mode of life. In this lecture we will examine into the details of these matters more particularly, and endeavor to discover their practical bearing upon the prevention and cure of cancer.

For the proper understanding of the relation of food and drink to cancer, and the satisfactory application of the principles involved, it is necessary to bear well in mind the chemistry of the body and the relation to nutrition of the various elements which contribute to form healthy and diseased tissues.

The human body is composed of some fifteen different elements, the relative proportions of which may be understood by the following table from Sherman, which represents probably as approximately correct an average as any that can be given.

COMPOSITION OF THE HUMAN BODY

	<i>Per cent.</i>
Oxygen, about.....	65
Carbon, about	18
Hydrogen, about	10
Nitrogen, about	3
Calcium, about	2
Phosphorus, about	1
Potassium, about	0.35
Sulphur, about	0.25
Sodium, about	0.15
Chlorine, about	0.15

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	<i>Per cent.</i>
Magnesium, about	0.05
Iron, about	0.004
Iodine	} very minute traces
Fluorine	
Silicon	

As the actual composition of the body is changing day by day, through the activities of the system, so that it is commonly believed that after some years all the tissues are entirely renewed, the daily wear and tear, as also the material expended in heat and activity, must be supplied by the diet. For the ordinary requirements of the system, in health, the appetite serves as a guide, which should suffice in man as in wild animals, to preserve the balance of nutrition. But man has also the power to *gratify the taste*, which must be recognized in our study as distinct from the satisfying of the appetite; and the refinements of civilization have added so greatly to the temptation of wrong and over-eating and drinking, as they have to many other temp-

tations, that it is questionable if reason, and what is often spoken of as the natural instinct for food, can be trusted in mankind.

It is to be remembered that the advance of civilization, and the facilities of transportation and cold storage, have brought from far and near an innumerable number and variety of articles for food and drink, including condiments, which bear no relation to the few simple articles formerly consumed; even the fruits which we eat are rarely ripened fully by nature, but are picked more or less green, and undergo an artificial ripening without the action of the sun, which is really akin to decay. In the combination and preparation of articles of food also, so-called civilization and refinement have made the greatest departure from the simple life of the aborigines, who are free from cancer, and with increasing ease and wealth throughout the civilized world more and more individuals are shar-

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ing in unnecessary and often harmful indulgences, more and more freely: and this is especially true of animal food, the consumption of which has increased so greatly. Many other elements likewise enter into the matter of the digestibility and consequent nutritive power of food and drink; such are nervous conditions, rapid eating, imperfect mastication and insalivation, heat and cold, character of the air breathed, micro-organisms, etc., and all the various causes which may derange the action of the digestive organs and so prevent the perfect metabolism between nutrient material and the cells of the body, as I tried to show you in some former lectures.

As is well known, the nutrition of man is supplied by the organic substances, protein, carbohydrates, and fat; these are found in various combinations in animal and vegetarian foods, and as a rule contain also much of the inorganic or mineral substances necessary for the system; all of

these with water, and its salts, and oxygen, supplied by the lungs, unite, through anabolism and catabolism, to build and maintain the human body in health.

It is also well known that in order to preserve health and proper weight there must exist in the economy a certain balance or equilibrium between the amount of the ingesta and excreta, representing the various elements which enter into nutrition; thus we speak of a nitrogen equilibrium, a carbo-hydrate equilibrium, a phosphorus equilibrium, and iron equilibrium, etc., some of which are disturbed continually in ill health and in various diseases, including cancer, as has been shown in our last lecture.

Until quite recently the principles of diet (even if not often carried out in practice) have been established on lines laid down by Carl Voit, of Munich; this eminent physiologist, after studying the dietary habits of various classes of workers, claimed that

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the adult man of 150 pounds, doing moderate muscular work, requires daily 118 grams of protein or albuminous food, 56 grams of fat, and 500 grams of carbohydrate, with a total fuel value of 3000 large calories, in order to maintain the body in equilibrium. But the remarkable and scientific experiments of Chittenden have demonstrated beyond question that perfect bodily and nitrogenous equilibrium can be maintained with one-third of the amount of protein called for by the Voit standard, and with a total value in the diet of only about 1600 calories, or about one-half of that indicated as necessary by Voit. These experiments were based on a group of five men of varying ages, professors and instructors at Yale, thirteen volunteers from the Hospital Corps of the United States Army, and eight students in Yale, all thoroughly trained athletes, twenty-six in all.

It would be quite beyond the scope of

this lecture to enter at all into the intricate questions connected with the metabolism of nitrogenous and other foods, but Chittenden has well put the reasons "why prominence is given to the establishment of nitrogenous equilibrium and why the proteid intake assumes a greater importance than the daily amount of fat and carbohydrate consumed. Fats and carbohydrates when oxidized in the body are ultimately burned to simple gaseous products, viz., carbonic acid and water. Hence these waste products are easily and quickly eliminated and cannot exercise much deleterious influence, even when formed in excess. . . . With protein foods, on the other hand, the story is quite different. These substances when oxidized yield a row of crystalline, nitrogenous products which ultimately pass out of the body through the kidneys. Prior to their excretion, however, these products—frequently spoken of as toxins—float through the body and may

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exercise more or less of a deleterious influence upon the system, or, being temporarily deposited, may exert some specific or local influence that calls for speedy removal. Hence the importance of restricting the production of these bodies to the minimal amount, owing to their possible physiological effect and the part they are liable to play in the causation of many diseased conditions."

When we consider the small share which nitrogen plays in the composition of the human frame, as shown in the table presented, only three per cent., it is easy to see how an excess of nitrogenous food must necessarily either pass off unassimilated or undergo imperfect metabolism, and so derange the general metabolism; and this is found to be the case in many conditions of disease, and, as has been shown, in cancer. Chalmers Watson and others have shown in a most remarkable manner, by animal experiments, that an

excessive meat diet alters very materially the microscopic structure of very many organs and portions of the body.

Beneke, who is often quoted, was one of the first to seriously consider the actual diet beneficial in cancer, his observations dating back to 1875 upon material in the service of Esmarck and Oldehop, who treated patients according to his plan. While the diet he gives is not wholly vegetarian, he limits the nitrogenous intake very greatly, and reported some very favorable results, with the complete disappearance of some malignant new formations.

Referring now to the data presented in the second lecture, in regard to the frequency and geographical distribution of cancer, we can understand better, on scientific grounds some of the reasons why cancer is so steadily increasing in civilized communities, and why in some sections of the earth it is less common, while certain

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aborigines have seemed to be almost immune.

We found that in England the per capita consumption of meat was 130 pounds per year, and that it had doubled during the past fifty years, while during the same period cancer had increased *four fold*; but in Ireland, where the meat consumption was estimated in 1895 at only 40 pounds per capita, or less than one-third that in England, the cancer death rate is very much lower, not much over one-half. We found that in Italy, where the per capita consumption of meat was the smallest of any European country the cancer death rate was almost the lowest. Also that in certain other countries, where vegetarianism was the rule, cancer was very infrequent, while among certain aborigines the disease was practically unknown; but we found also that in both the latter classes of individuals cancer has slowly increased, in proportion as the inhabitants of different

sections had come under the influence of modern civilization, and adopted the dietary and other habits of foreigners.

In the United States cancer has certainly increased very greatly during the last fifty years, and statistics were presented showing that in seven of the largest cities, during the past five years, the number of cancer deaths had augmented between seven and eight per cent. It is known that the consumption of meat here has increased steadily, until in a communication from the Bureau of Agriculture in Washington we learn that it had recently reached the enormous amount of 172 pounds per capita yearly, much more than in England.

Cancer has been repeatedly spoken of as a disease of civilization, and there are many other elements besides meat to be considered in connection with its etiology.

Coffee and tea are so widely and almost universally used in civilized countries, and their apparently pleasant effect is so great

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that few realize the harm that may result therefrom; although from time to time their injurious effects, especially along the line of digestive and nervous troubles, are dwelt on by medical writers. Of late years, however, more attention has been paid to their influence on metabolism and also to the relation of their consumption to the increase of cancer. From a report to the House of Commons in England, Holland is shown to be the largest per capita consumer of coffee of any country in Europe, and the cancer death rate there in 1905 was among the highest, while Hungary was the smallest consumer of coffee, and the cancer mortality in 1903 was only 39 per 100,000, or a little over one-third that in Holland. It may be interesting to know that Thompson states that "the people of the United States consume one-third of the total coffee produced, or more than Germany, Austria, Hungary, France and the United Kingdom combined. On the

other hand England and her colonies consume one-half of the world's output of tea, and the United States but one-fifth of it."

The scientific basis of a possible relationship of the consumption of coffee and tea to the prevalence of cancer may be better understood when we remember that caffeine and theine belong to the xanthin group, and contain exactly the same equivalent of nitrogen as uric acid. A single cup of coffee of fair strength, it is stated by Hutchinson, contains from one to three grains of caffeine, and a cup of fairly strong tea 1.21 grains, or more than the average medicinal dose of this drug; and all know how great may be the consumption of coffee and tea by some individuals, and that many of the working class, especially, consume enormous amounts of tea, which is kept brewing all day. Roberts has very clearly demonstrated, by clever experiments, that tea interferes very

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greatly with both the salivary and gastric digestion.

Alcohol, or some of its combinations, has also been shown by several observers to be undoubtedly an element contributory to the causation of cancer; this relates not only to countries or cities where the consumption is the greatest or least, but also to various occupations, in which statistics show the more or less abundant use of distilled or fermented drinks, and deaths from the same, and in regard to total abstainers; and a careful study of the subject makes it pretty clear that the incidence of cancer corresponds in a measure with drinking habits; that is, that cancer mortality is highest among those classes of persons among whom primary or secondary mortality from alcoholism is greatest. There are so many elements to be taken into consideration in connection with the derangement of metabolism which leads to cancer, that it is difficult to fix the precise

influence which each may exert; but in watching cancer cases for any length of time it is easy to see the harmful effect when alcoholic beverages are indulged in, and the improvement when all such are absolutely excluded.

An interesting confirmation of the beneficial results of a low diet and simple life, as regards cancer, is found in certain reports of Commissioners of Prisons and Asylums in England, where the matter has been studied, as given by Russell. "Asylums contain an excessive number of persons who have inherited or acquired constitutional weaknesses, and in many cases tendencies toward consumption or cancer; also many alcoholics who are prone to these maladies. Yet the habits and rules of these institutions reduce the cancer rate much below the rate of the classes from which they are drawn, and below the rate of both occupied and unoccupied persons." The same is observed in regard to many

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religious orders, where the members lead a very simple and frugal life, and where cancer is reported to be almost unknown.

Kessler has called attention to the disturbance of sulphur partition in cancer in connection with diet, and the desirability of excluding those foods exhibiting an excess of sulphur, giving lists of the same and indicating a satisfactory diet.

Packard has made a strong argument in regard to the value and importance of the mineral elements contained in plant life, in connection with the disturbances in these same elements which has been observed in connection with cancer, as we have already seen. He recalls that modern chemistry teaches that the inorganic principles of the vegetable kingdom are absolutely necessary to the highest degree and type of animal tissue and health, and resistance to disease. Plant life is the connection between the minerals and salts of the earth and animal life, but in the

manufacture or refinement, and cooking, of products of the vegetable kingdom, many of them are largely demineralized; this especially illustrated in the case of fine white wheat flour, rice, potatoes (in peeling and cooking), etc. So that while animals get plenty of mineral matter from plants and the earth, man gets but little, and while the herbivorous animals are rarely affected with cancer, civilized man is succumbing to it more and more. It is stated that among savage tribes, who are practically free from cancer, the water in which vegetables are cooked is also consumed as food, thus securing all the salts. The same idea has been popularly presented to the public in a startling manner by McCann in a book which, with a great deal of verbiage contains a large amount of valuable information concerning nutrition, and its disturbance by erroneous, or worse, preparation and administration of food.

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Possibly there are other dietary elements which may play some part in the causation of cancer, but the demonstrated facts in regard to them are so few and uncertain that they need not detain us here, although it is certainly desirable to investigate any that seem to have reasonable support.

Some of these which have been suggested probably have to do with local irritant action on the digestive organs, as we have previously seen that local irritation undoubtedly plays an important part in the determination of the actual time of occurrence and site of the cancerous disease. Thus, some have ascribed cancer to hot food or drink, or to stimulating drink, condiments, etc. It is quite possible that these contribute to the development of cancer in the pylorus, irritating the secreting cells in their passage. Mayo says: "In civilized man one-third of all cancers are seated in the stomach. This is not known

to be the case in uncivilized man or in animals. There should, therefore, be something—some one cause—which causes this preponderance. The acid secretion may favor its development, for when we come to the colon, also with an acid secretion, we again meet with cancer, and we seldom see it in the alkaline, small intestine. Gastric ulcer, which may be precancerous, is connected with hyperacidity.” In Scandinavia cancer of the stomach is remarkably frequent, according to Soëgaard, thus, of 1235 cases in Norway, 73.9 per cent. were in that location. In our last lecture we found that cancer in general was connected with a lowered alkalescence of the blood, and all our studies show hyperacidity to be related to cancer genesis; and nitrogenous acidity, or uric acid (purin, xanthin, etc.), undoubtedly plays a great part in inducing malignant action in tissues, as Haig has so long contended, even in regard to cancer.

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The increasing frequency of cancer of the mouth, œsophagus, and stomach in men certainly looks toward an irritating character of substances which traverse these regions, including alcoholic drinks, and the irritant effect of tobacco should not be overlooked in regard to mouth lesions. But of the millions who use tobacco only very few are affected with cancer, and only those who are predisposed thereto by some metabolic disturbance, whose true character and other manifestations are not yet fully determined. We have already referred to the practice of so-called betel chewing in the Far East as a frequent cause of cancer within the mouth, also to the wearing of the Kangri charcoal baskets in India, for warmth, causing a burn on the front of the body which may develop into epithelioma; it is claimed, however, by some that of the many cases of "Kangri burn," but few result in epithelioma.

There are yet other considerations con-

cerning the relation of diet to cancer which are worthy of attention. We have mentioned some principal agents, which seem unquestionably to have an influence in the production and continuance of cancer, namely, proteids, coffee and tea, and alcoholics. But millions of human beings partake of these with apparent impunity, while in the relatively few they appear to have cancer-genetic powers. This need not surprise or puzzle us any more than do the many other problems in medicine which we are seeking to solve: for we know how often it happens that the system reaches a point where certain things, once well borne, are no longer tolerated. We know, for instance, that Port and Madeira wine certainly can cause gout, but with many individuals they may be indulged in freely for some time before this result follows: likewise that tobacco may even be abused for a long time, without apparent ill effects, when suddenly there is a revul-

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sion of the system and the slightest use of tobacco will be intolerable: also that many edible substances which have long been well borne, will at a certain time act unfavorably and excite eruptions, urticaria, acne, eczema, etc.

Psoriasis also furnishes an illustration which may be of service in understanding the relation of diet to cancer; for psoriasis is characterized by a disordered epithelial growth, which both shows on the surface and manifests itself by epithelial prolongations into the corium, which are quite comparable to the ingrowing cellular masses of early cancer: moreover cancer is not very rare in psoriasis patients. In this eruption it has been very clearly demonstrated, clinically and experimentally, that error in nitrogenous metabolism is commonly at the bottom of the eruption, which has been seen to promptly disappear entirely simply under an absolute vegetarian diet, correctly regulated, excluding also

coffee and alcohol, without the use of any medical treatment whatever, internal or external; but, of course, this result cannot always be obtained, and sometimes the eruption will relapse during what is thought to be a strict vegetarian diet. There must, therefore, be some systemic disturbance which causes nutritive material, at some particular time, thus to derange cell action in the eruptions mentioned, and the same is true in regard to the production of cancer.

Some years ago Braithwaite called attention to the occurrence of cancer among certain peoples who were vegetarians, and attributed it to the great amount of salt which they consumed. While the suggestion of salt being a cause of cancer has been ridiculed, it is quite possible that a great excess of sodium chloride may disturb the salt equilibrium in the blood, by replacing the potassium which is so necessary for proper cellular nutrition, and also

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by hindering the excretion of uric acid, as Haig has pointed out.

When we inquire into the cause of the systemic disturbance which tends to such faulty metabolism that the nutrition of cellular structures is deranged, even to the degree of taking on malignant action, we find many possible elements, more or less connected with what is known as modern civilization, to which we have time to but briefly allude. Williams has shown pretty clearly that wealth, with its tendency to luxury and idleness, greatly increased the proclivity to cancer: not only is this observed in different countries, but in certain cities the difference is very striking between the cancer mortality in sections which are occupied by the rich and well-to-do, and those in which the poorer classes are herded. Also in England it was found in one decennium that cancer mortality was more than twice as great among the well-to-do men, having no specific occupa-

tion, as among occupied males in general, the ratio being 96 to 44.

Change in the mode of life, and sudden changes of environment have also been found to have a great effect in the production of cancer, as has already been mentioned in another lecture.

Finally, for our time does not permit a fuller discussion, nervous conditions unquestionably can and very often do exert a profound influence on the secretion of the various organs of the body, and can so disturb digestion, metabolism, and nutrition that the most varied results may follow, to which the names of different diseases are given; so that nerve strain, more or less incident to modern life, must be accredited with a certain share of influence in the production of cancer. The part which imperfect and deficient intestinal and urinary excretion play in inducing or perpetuating the disease will be treated of in later lectures.

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In our next lecture we will consider certain matters relating to the medical treatment of cancer, but from long experience and study I am firmly convinced that such measures are of relatively little service unless coupled with a rigid care of the diet and hygiene. As Bell remarks, "Cancer is essentially a disease supervening upon a persistent neglect of hygienic laws." It is a disease of sub-oxidation, and all the hygienic elements of importance in tuberculosis are equally necessary in cancer; fresh air and sunlight with, as far as possible, an ideal regulation of life in all its aspects, are indispensable.

Inasmuch as there is no specific medical antidote for cancer, and we cannot be certain of securing at once the metabolism of health, it is wise to remove and to keep away from the system those articles which have been shown to have more or less influence in its production, namely, animal products, coffee, tea, and alcoholics. Time

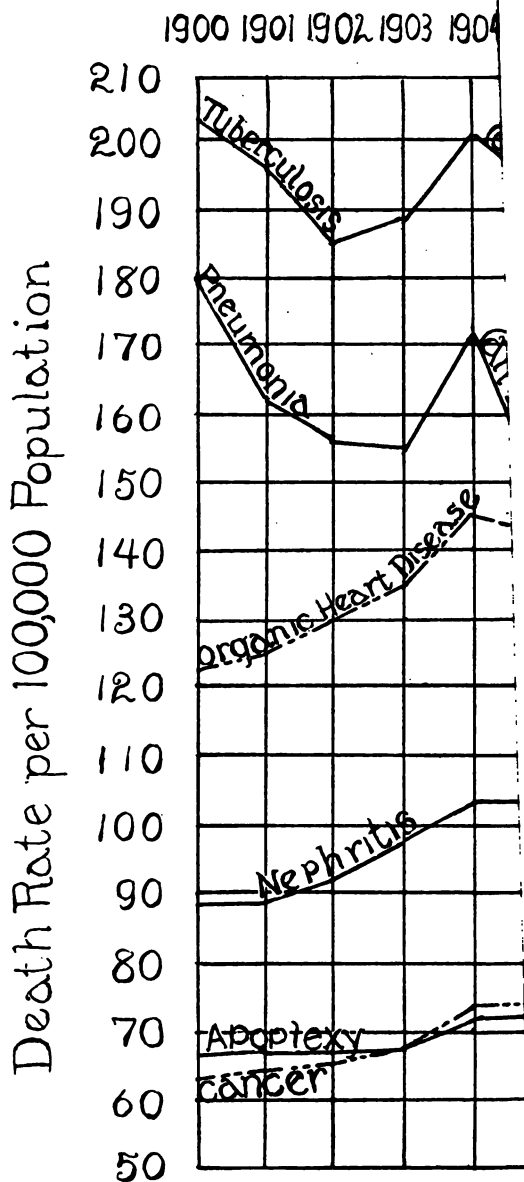
does not admit a discussion of vegetarianism, nor is it necessary, for there is abundant evidence in literature and on every side that perfect health can be maintained thereby, and I went over the subject pretty thoroughly two years ago. The vegetable kingdom contains protein enough to build up and maintain the tissues of the body, as is shown in animals, and an extra number of calories can be easily obtained from butter, of which a quarter of a pound contains some 800 calories, or fully one-third of the total daily quantity required by most individuals. The details of a purely vegetarian diet, which experience has shown to be of the greatest value in cancer, can be worked out for individual cases as required. I am also firmly convinced that in this absolutely vegetarian diet, with other proper hygienic and medicinal measures, to be detailed next week, we shall find a great power for the prevention of cancer; although many persons are already so

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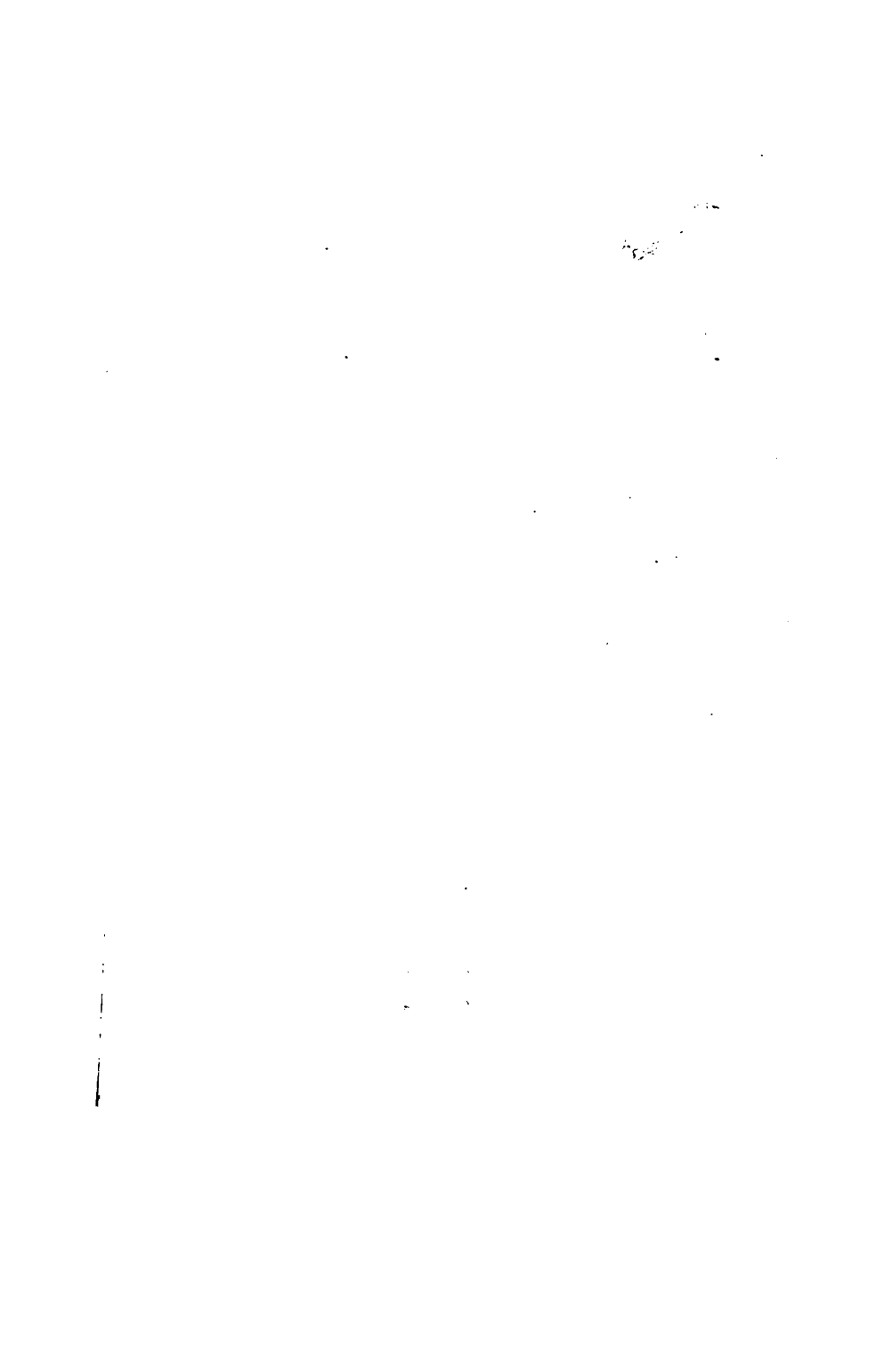
saturated with poisoned blood and tissues from prolonged errors of life, that perhaps no very striking general effect on the community can be obtained therefrom in this generation.

In closing this lecture I must again urge upon you the necessity of great patience and perseverance, with very much careful study of the patient in all particulars, over a great length of time, if really favorable results are to be obtained in cancer; and this is true whether the disease be incipient, or fully developed, or even post-operative. The causes of cancer are deep seated, and, as with many chronic affections, there is no tendency to continue an improvement once begun, but under a return to the same conditions as before the disease will certainly assert itself. We have learned the lesson of assiduous perseverance in tuberculosis; let us learn it in regard to cancer.

From United States



The mortality from organic heart disease, nephritis, we accept the fact that the increasing death rate, especially from erroneous eating and drinking.



LECTURE V

MEDICAL TREATMENT OF CANCER

It is remarkable that so little serious attention has been paid to the medical treatment of cancer, in standard works, in view of the occasional strong statements and reports which have appeared from time to time in current literature and in occasional books, dating back for many years. Much of this, of course, relates to imperfect observation and erroneous diagnosis, and also to crude medical knowledge, but there have been also plenty of good men, who knew the disease and have reported favorable results, and even the complete disappearance of cancer, under dietetic regimen and proper medication alone, without operative interference of any kind.

Not to go back too far, reference can be

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made to Lambe, who one hundred years ago wrote clearly in regard to the causation of cancer from luxurious living, and adduced strong proof to show the effect of diet in curing certain cases of undoubted cancer of the breast and uterus, the diagnosis of which was confirmed by prominent surgeons of the day, several of whom endorsed the vegetarian diet. Abernethy wrote pointedly, soon after, regarding the constitutional origin of tumors and says, "There can be no subject which I think more likely to interest the mind of the surgeon, than that of an endeavor to amend and alter the state of a cancerous constitution. The best timed and best conducted operation brings with it nothing but disgrace, if the diseased propensities of the constitution are active and powerful. It is after an operation that, in my opinion, we are most particularly incited to regulate the constitution, lest the disease should be revived or renewed by its disturbance."

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He then gives an endorsement of Lambe's dietetic treatment of cancer, and presents several reasons why it should be fairly tried.

In the classical work of Walshe we find numerous references to the constitutional nature of cancer, original or quoted from recognized authorities, as well as expressions in regard to the futility of expecting that surgical interference would cure the real disease in any great proportion of cases. He says, "It would in theory appear that the removal of a tumor cannot in itself cure the disease, as the local formation is but a symptom of a general vice of the economy." . . . "This tissue being, as the normal textures, the seat of nutrition, is like them susceptible of its disordered actions," and he alludes more or less to the effect of diet on the disease.

The late Willard Parker, one of New York's great surgeons, in a study of 397 cases of cancer of the female breast, ob-

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served from 1830 to 1880, wrote very strongly in regard to the constitutional relations of cancer. In considering the etiology he places constitutional causes first, and says, "luxurious living, and particularly excess in animal food, increases the waste products of the body, and if coupled with insufficient exercise, the waste products are retained in the system and have a tendency to produce abnormal growths." . . . "Cancer is to a great degree one of the final results of a long-continued course of error in diet, and a strict dietetic regimen is, therefore, a chief factor in the treatment, preventative and curative." Concluding his study he says, "There is such a *consensus* of opinion as to the advisability of early removal of the growth, that a discussion of the subject would be useless. So then, in the first place, let us remove the tumor, and thoroughly. But after we have done so, after we have taken it out by the very roots, is this sufficient?

No. We must then adopt the means stated above to prevent a second development. We must change the diathesis; we must seek to modify the patient's constitution so that it will be no longer prone to reproduce the disease: and then only may the surgeon be satisfied that he has done his duty." He further says, "In regard to the effect of abstemiousness on cancer I can speak with great positiveness, that vegetable, or at least a very bland diet, does check the progress of the disease, and, in some cases now under treatment, has been attended by an alleviation of symptoms; and in a few instances even by a recession of the growth." He also quotes from Sir Astley Cooper some strong language which, as he says, "shows a broader and more enlightened view of the subject than is contained in the writings of some more recent observers, who have supposed that they were working far in advance of the great English surgeon"; the rather

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long quotation then given proves that that great surgeon also believed absolutely in the constitutional nature of cancer.

Sir James Paget is often mentioned by writers, sometimes even with derision, as a believer in the Constitutional Nature of Cancer, and his words are very strong; says he, "I believe it to be constitutional, in the sense of having its origin and chief support in the blood, by which the constitution of the whole body is maintained," and speaks at some length in his lecture, in support of this view; I will give you one other small quotation. "The existence of the morbid material in the blood, whether in the rudimental or in the effective state, constitutes the general predisposition to cancer," etc. It is remarkable that the views of this prince of surgeons and pathologists should not have carried more weight in regard to cancer, when his other views are so well accepted.

Time does not permit us even to refer

to the great mass of more or less similar views and corroborative evidence scattered in books and current literature during the last fifty years, but a few more references may be given. In 1884, and again lately, Merriam has called attention to cancer occurring as a reversion of tissue cells to earlier or embryonic forms under the influence of a disordered, or as he calls it, a poisoned blood stream, from over nutrition from a meat diet; and Haig has long maintained that uric acid is a prominent factor in the etiology of cancer.

While few will agree with Haig in all his conclusions, there is no doubt but that a faulty nitrogenous metabolism plays a not inconsiderable part in the causation of many diseased conditions, as has been also shown in a previous lecture to be true in regard to cancer: and while it may not be actually uric acid or urea which does the harm, as these are relatively harmless end products of metabolism, it is true that the

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purin and xanthin bases, of many forms, and imperfectly oxidized nitrogenous elements, are active agents in the production of disease. Venus and Isenberg have both written very strongly in regard to the unquestioned value of a vegetarian diet in cancer, the former citing many supporters of the view, from ancient times to the present. If time permitted any amount more of corroborative evidence could be given in regard to the connection of cancer with metabolic disorders.

There have been many scattered articles concerning the chemico-pathology of cancer, all looking in much the same direction as that which we are pursuing, which cannot even be alluded to, but brief mention must be made of a most illuminating and suggestive study by Bristol. Reviewing the many theories of the etiology of neoplasms he shows pretty clearly that cell reproduction comes from outside the cell, or from the surrounding cell medium by a

process of diffusion, or by a definite chemical attraction, and is closely associated with the nutrition and growth of the cell, especially the nucleus; also that a fixed ratio between the salts in the blood, lymph, and tissues is necessary for normal activity and reproduction of cells; further that a disturbance in this ratio and an upset in the chemical equilibrium will lead to abnormal metabolism, growth, and reproduction, and result in an atypical growth in the local area involved.

As before remarked, it seems strange that the medical profession and the public have been so slow in accepting the views here presented. But both have become so obsessed with the idea that nothing can be done for cancer except operative procedure of some kind, and so dazzled with the glamour of modern surgery, so fascinated with the apparent definiteness of laboratory proceedings, and so attracted by the glory of purely scientific research,

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that it is very difficult for any other doctrines to make headway: moreover, any dietary or medical treatment, prophylactic or curative, is very tedious and also difficult, in the way of attention to minute details, etc., and patients weary of restrictions and are restless at seeing little or slow progress made. Haig found the same difficulty with Hospital patients, but does not hesitate to state that in private practice "some undoubted cases of cancer have gotten well" under his dietary restrictions.

But when it is remembered that it is calculated that, under present existing conditions and treatment, fully 90 per cent. of those attacked with cancer ultimately die from it, and that there were last year nearly 50,000 recorded deaths from this cause in the "Registration area" of the United States, covering but two-thirds of its population, and that the disease is rapidly and surely increasing, it behooves the Medical Profession to put forth the best

efforts possible to stay the progress of the disease. In New York City, according to the Weekly Bulletins of the Board of Health, there were from May to November, 1914, in 26 weeks, 2173 deaths from Cancer, Malignant Tumor, or an average of 83.5 per week, almost twelve deaths daily from malignant disease.

Operative surgery has unquestionably been of great service in certain cases and along certain lines of cancer, and the claim for the earliest possible removal of morbid growths is certainly a just and proper one; moreover, with our present knowledge it is perhaps wise to attempt to remove the offending mass while there is some hope or prospect of benefit, and personally I am occasionally advising this in proper cases. X-ray and radium have also their sphere of usefulness in a certain class or group of cases, but the excessive exploitation of the latter, not long ago, did harm in raising false hopes in multitudes of sufferers, and

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as these were shattered so also was confidence shaken in regard to all help for cancer from other than surgical removal.

But every one who has been brought much into contact with cancer knows full well what a mass of recurrent cases exist, possibly many of them due to bad operation, in regard to which surgery has to acknowledge that it can offer nothing more, that they are inoperable and therefore they are regarded as incurable. Every one also knows how many cases there are which have advanced so far before coming to a competent surgeon, that they are also inoperable. Every one likewise knows that there are numerous cases of cancer in internal regions, and in certain localities, and presenting peculiar features, which are also recognized as inoperable, at least with any proper justification. Thus it may safely be said that of the total number of cases of cancer existing at any one time, which would be included under the above classes,

fully 50 per cent. are such that operative surgery can offer no hope of material benefit: also, it must be acknowledged that but a relatively small proportion of all cancer cases are likely to secure the very best surgical service, such as claims the highest percentage of success.

For this large number of hopeless victims of this dire disease, at least, proper dietary and medical treatment should be most carefully studied and patiently applied, with the hope and expectation that the same, if correctly employed, would more or less hinder or check its progress or prevent a recurrence after operation. But experience shows that such measures, if taken promptly and thoroughly, can also prevent the development of early threatening lesions into those of malignant character, suggesting surgical removal, and these should never be neglected when there is the slightest suspicion of cancer. For it must be acknowledged that the surgery of

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cancer is only an attempt at the physical removal of a something, which medical science and art should not have allowed to develop out of normal tissue. And in the matter of the general prophylaxis of cancer it is certainly worth while to consider and act strongly upon the facts which have been presented, especially in those individuals who have any hereditary suspicions, or who exhibit the habit of body or life which could lead to the development of cancer.

The medical care of cancer, as of all diseases, naturally includes both prophylaxis and the treatment of a morbid condition which has already developed; the former of these is always considered to be of the highest importance.

Prophylaxis of Cancer. From what has preceded in this and other lectures it can readily be understood that the prevention of cancer lies largely along dietary and hygienic lines, including such medical atten-

tion as shall secure healthy action of the secreting and excreting organs. If the figures and facts heretofore given are correct, if cancer has surely been observed to increase under certain conditions of life, if it is found to be absent or relatively rare under certain other conditions of living, if cancer has been known to disappear spontaneously in certain individuals, especially when they have changed their mode of living, it must then follow that when the exactly correct habit and state of life are continually assumed the disease will not occur.

In other words, when the blood stream nourishing the tissues is ideally correct, the individual cells of the body perform their functions normally, and as each cell is worn out it is removed by a healthy catabolism and renewed by a normal anabolism, and homologous cells then replace those which have ceased to be able to perform their function. On the other hand,

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when there is perverted metabolism we have various forms of deranged action, to which we give the names of various diseases, one of which is cancer: here in place of homologous cells maintaining the tissues in a normal condition, we have a malignant metamorphosis into heterologous cells, which take on a disorderly or rampant action, and refuse to assimilate themselves to others, so as to form healthy tissue; we have then a mass of low vitality, tending to easily break down or ulcerate, and to extend their malignant action to adjoining tissues, under the continued influence of a contaminated blood current. When once ulceration has occurred, the ordinary parasitic micro-organisms, which are practically omnipresent, find an acceptable nidus, and aid in the destructive work, and also in the systemic poisoning of the tissues.

The aim, then to be sought in the prophylaxis of cancer is to secure the most

perfect blood stream possible, which is accomplished by simple living, perfect mastication, and the avoidance of excesses, especially along the lines of protein, tea and coffee, alcohol, etc., with the maintenance of healthy habits of life, especially in regard to bowel action. As already intimated, we do not know just why in some persons and at a certain time a disturbance of metabolism results in perverted cell growth, but there is, of course, a reason, and the physician's duty is so to regulate the patient's whole life that the metabolism is kept in the state of perfect health. All this seems very simple and trite, but really it is a task which is most important and often most difficult to accomplish, and requires very careful study and infinite patience. Dr. Kellogg, of Battle Creek, says that of the many thousands who have passed under his care he has not known cancer to develop in any one who had been faithful to the principles which he incul-

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cates, and I may say the same of the many who have been under my care in private practice, often with notes extending over many years.

Time does not permit of going into detail here in regard to the special features of diet and hygiene, which I discussed with you fully two years ago, in my lectures, which were subsequently published: suffice it to say, as previously remarked, that the refinements of civilization add greatly to the temptation of over-eating, and wrong eating and drinking, as they do to numerous other temptations, and in many instances it requires the utmost amount of grit as well as patience, both in the patient and doctor, to secure the perfect result necessary.

It is understood, I trust, that I believe that for the proper treatment of cancer, and to prevent its occurrence and recurrence, it is absolutely necessary to maintain a perfect vegetarian diet, which in-

cludes even the exclusion of eggs and milk, with food; but the yolk of eggs may sometimes be taken with advantage, and also milk alone and separate, at a body temperature, one hour before eating, according to the plan which I have often demonstrated to you. As remarked in my last lecture, the vegetable kingdom contains proteid sufficient for the system, and the diet should be so arranged as to give the requisite quantity of calories, in proper proportion, of vegetable protein, carbohydrates, and fats: the latter is accomplished largely with butter, of which a quarter of a pound may be taken daily. Many writers on diet hold that the protein in casein acts differently from that contained in meat, and this is allowed in many vegetarian dietaries: it also contains phosphorus, which is an integral part of living cells, especially their nuclei. When nutrition seems to demand it, this can, perhaps, be added to the diet, mixed with cereals or in soups, and a small

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amount of certain kinds of cheese is also sometimes allowable. It is impossible to elaborate the details of diet fully in a lecture such as this, and I must refer you to the many excellent works on food and nutrition (Hutchinson, Sherman, Thompson, Buttner, Braithwaite), for further information.

The *medical treatment of cancer patients* is a matter which should receive most careful and constant attention; indeed, the patient in whom cancer is threatening or has developed should be under the watchful and unremitting guidance of a physician who grasps the importance of the matter and is prepared to guard the health, and to meet and correct functional disorders as they arise; for a careful study of patients with cancer for many years has constantly shown me gross errors in their life and in the action of various organs, and consequently disorders of metabolism

which are of weight in connection with the disease.

The first point of importance to which I wish to call your attention is in regard to the action of the bowels. Some of you may remember that even that great surgeon, Sir Arbuthnot Lane, has recently emphasized the fact that one of the terminal results of intestinal stasis may be cancer. This may seem strange, but it would surprise you if you knew how very commonly there was imperfect intestinal excretion, or intestinal stasis, as it is now called, in these subjects; indeed, I might almost say that it is the rule, and that this assuredly plays a most important part in the auto-intoxication which leads up to cancer; in fact, I almost feel like saying that the toxins produced by the millions of micro-organisms generated through intestinal stasis and fecal putrefaction are the real, incidental cause of cancer. Under a perfect vegeta-

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rian diet there is less likelihood of trouble in this direction, but even then there is necessity of watching continually, that there may be at least, one, full, free movement of normal character, daily, after breakfast. In many cancer patients I have secured this by having the entire potato eaten, skin and all, whether boiled or baked; by this means also one gets the full value of the inner layer of skin, which contains the mineral salts so necessary to the economy, which are entirely lost when the potatoes are peeled before boiling; so that potatoes should always be cooked in their skins, even if the outer layer is stripped off afterwards.

But the regulation of the bowel action should not be left to the judgment and action of patients, with a spasmodic use of remedies, and with alternate constipation and purgation; the physician himself should carefully guide and direct the proper treatment frequently enough to se-

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cure perfect results. Of course each one may have their own ideas and methods, but I have long used with the best results the remedies which you have often heard me order in this clinic. Thus, at the beginning of treatment, and often on the same days of many succeeding weeks, I give ℞ Ext. Colocynth. Co., Mass Hydrarg aa gr. x Pulv. Ipecac gr. ij M. Div. in Capsules No. IV. Sig. Take two at night and two on the second night after. For continuous use, or in the nights between these, I have long used a compound Cascarin tablet (℞ Podophylin, Aloin, Cascarin aa gr. $\frac{1}{4}$) one or more each night, or have depended on the Cascara in a mixture of which I shall shortly speak. I do not like the action of salts or laxative mineral waters in these cases, and, of course, enemata are entirely useless to secure real, effective liver and intestinal action, and should be used only in an emergency.

The action of the *kidneys* is also always

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a very important element to watch and control in cancer patients; this does not have reference to albumen and casts, or glycosuria, but to a functional derangement of the secretion: for the former are relatively infrequent compared to the latter. This is so large a subject that time does not permit of its elaboration; I made the matter pretty clear to you in my lectures not long ago on the relations of diseases of the skin to internal disorders. I must tell you, however, that, as a result of numberless studies of the urine of many cancer patients, I have almost constantly found a deficient or defective urinary secretion: in many cases I have had the total daily amount accurately measured, and reported in writing each week over long periods, and while the total quantity may at times be near normal, the total solids sometimes fall to, and remain at, even less than one-half the amount which should be passed for the patient's body weight.

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I want here to urge upon you the importance and value of repeated volumetric examination of all possible ingredients of the urine, as indicating in the best manner obtainable the state of the arterial blood, from which the urine is derived: especially is this true of the exact volumetric acidity, representing the diminished alkalescence of the blood common in cancer.

The actual medicinal and dietary treatment of the various possible departures from normal can hardly be elaborated here, but they are along very much the same lines which you have often watched in connection with certain diseases of the skin; for you must remember that carcinoma is an epithelial disease, and that the laws of nutrition are much the same for this as for various other morbid conditions of the economy. I may say, however, that very many of my cancer patients have received the greatest benefit from acetate of potassa, often in the formula familiar to you (R

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Potass. Acetatis ʒi Tinct. Nucis Vomic ʒiv Extr. Cascar. fld. ʒij-ʒiv Extr. Rumicis radicis fld. ad ʒiv M. Sig. One teaspoonful half an hour before eating, well diluted). For many years I have administered potassa largely to these patients, and it is gratifying to find not only support but an incentive to its greater use, in the studies and experience of Ross, who pushes it to a very great extent, giving as high as 240 grains of potassium phosphate in a day. Ross also advises the free use of crude or brown sugar, as it contains a large proportion of potassium salts, which are for the most part removed from the white or refined article. I must also remind you of the great value of Bethesda water, in washing out the kidneys, given a tumblerful with each meal, and one tumblerful, hot, one hour before breakfast and also an hour before the evening meal.

The *blood* should also be watched, and in my hospital cases I have a count made

every week; for the hæmoglobin content, and the number of the erythrocytes, and the percentage of the varieties of leucocytes afford very valuable information as to the physical state of the patient, and the progress or recession of the cancer, and in a measure these data are of value in directing therapy. I have also the *weight* of the patient taken and recorded each week, as a guide to nutrition and dietary matters. Also the *saliva* is tested and recorded before and after each meal, and furnishes an indication in regard to the administration of alkalies.

Iron is found to be greatly wanting in the blood and tissues late in cancer, and should always be an element in the treatment of this disease. Skene Keith, noticing that after a cancerous mass was removed the blood recovered in regard to hæmoglobin and erythrocytes, administered iron with arsenic, with the greatest benefit, the growth shriveling up and the patient

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gaining in weight; he recommends the citrate of iron and ammonia, the preparation which I have given to patients for many years past. He also advises hypodermic injections of iron, arsenic, and soda, and reports cases thus treated, some of them with good results. There is a loss of phosphates in these patients, and phosphate of iron or other phosphates are valuable.

When considering the metabolism of cancer mention was made of derangement in the action of the ductless glands in this disease, and of the use and value of certain preparations of the internal secretions in its treatment. While a hormone therapy of cancer is still in its infancy, in spite of many books and innumerable journal articles on the internal secretions, it is quite possible that the future may demonstrate its efficacy in helping to control the disease: for, that the latter are efficient agents in carrying on the vital processes in the

body, there can be no question, although as Biedl remarks, "Our knowledge of the chemistry of hormone formation is very slight."

But considerable clinical evidence has been accumulated that one at least of the internal secretions, namely, that of the *thyroid*, has a definite beneficial effect upon cancer; this I have administered to many cancer patients, and have reason to believe that it contributed to the favorable results obtained. Sajous, believing that insufficiency of the adrenal secretion is an element in altered nutrition, advocates thyroid extract on the ground that it stimulates the activity of the adrenals: Vincent quotes Erlich as believing that "there may be substances circulating in the organism which may stimulate the body cells to resist the athreptic influence of cancer cells." Shirlaw advises a tablet made of thyroid 6 grains, supra-renal $\frac{3}{4}$ grain, and pituitary $\frac{1}{16}$ grain. While the treatment by

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these hormones from internal secretions is really in rather an experimental stage, it would seem more than probable that the correct use of these pluriglandular extracts, in combination with other proper dietary and medicinal treatment, would be of service in the prophylaxis and treatment of cancer. I have been a little cautious in the use of thyroid, beginning with two or three grains with meals, three times daily; for, as a promoter of catabolism it can disintegrate tissue faster than the emunctories can remove the effete products, and may thus poison the system.

Serum- and Vaccine-therapy are yet in a stage of development, and as I have had little or no personal experience with them I will not attempt to discuss their value, which time alone will demonstrate. While there have been many claims of success from treatment along these lines, unfortunately they have not been confirmed by later observations by others: I may say

frankly that they have never appealed to me as rational, in consideration of the views and experience I have long had along other lines of thought and practice. If, however, future investigations shall confirm some of the remarkable reports which have been made, the latest of which is by Nowell, there may be opened a non-surgical line of treatment of cancer which would mean much to many otherwise hopeless cases.

The *local medical treatment* of cancer is also an important matter which should receive careful attention. For many years in early cancer of the breast, under proper, general, dietetic, hygienic, and medicinal treatment, I have seen the lump vanish under the continued application of the iodide of lead, in Hebra's diachylon ointment of the German Pharmacopœia (3ss-3i ad 3i), and in very many instances I have had the assurance of patients that its application relieved the pain and was

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of the greatest benefit also in axillary and other metastases. In other cases the continuous painting with ichthyol (50 p.c. in water) has seemed to be of great service, in combination with other proper treatment: all of this in earlier cases, before ulceration has occurred.

After ulceration has set in, either in primary or recurrent cases, proper local medication is very important. The ordinary micro-organisms, finding a suitable nidus, very readily germinate and add greatly to the suppuration, and consequent exhaustion and pain, and by their toxins, aid also in the general contamination of the blood and tissues. Hydrogen peroxide, laid on with pledgets of absorbent cotton is often of great service in changing the conditions of the part, and when followed by Russian oil, also applied with cotton, makes a most satisfactory dressing. Adrenalin 1-1000 is also often of service, both in checking active hemorrhage, and in relieving pain,

and shrinking up the granulations. Ichthyol, 10 to 25 per cent. in water, and permanganate of potash in water, 1 to 2 per cent., are also often of service, likewise acetate of aluminium, 3 to 5 per cent.: methylin blue and fuchsin in watery solution, 1 to 2 per cent. were formerly much used, and often served a good purpose. It is understood, however, that none of these measures have any great curative effect on cancer, but as contributory elements they should never be neglected. Ichthyol ointment (℞ Ichthyol 3ss-3i Zinc Oxid 3ss Unguent. Aquæ Rosæ ʒi) often proves most satisfactory, spread on thin layers of absorbent cotton, and changed several times daily, after soaking the part with one of the liquids above mentioned.

Time does not permit us to go into the matter of the X-ray, which undoubtedly has often been of great service in certain cases of cancer: it should be carefully but systematically employed, once or twice a

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week in connection with other medical treatment; radium can also be of real service in these cases. In some inoperable cases a mild surgery, especially with the actual cautery, in the way of removing exuberant masses, which would have to slough off, can add much to the good progress of the case, and the occasional excision of cutaneous nodules removes just so much cancerous tissue, which by its secretion would poison the system.

In our study thus far we have seen that there is much to be done for cancer besides the surgical removal of the offending mass, the existence of which is an opprobrium to medical science, which has not thus far guarded mankind against it: and until time has passed, and by right living and proper medical attention cancer has ceased to be produced, or become greatly diminished, surgery will probably be a great factor in its treatment, as its mortality statistics are constantly improving. But there can be no

question but that with the adoption of proper dietary and medical treatment, even from the first suspicion of a malignant growth, and before, as also after operation, the final results of operative procedure will be even far better than have been yet attained.

LECTURE VI

CLINICAL CONSIDERATIONS AND CONCLUSIONS

THE test of everything lies in the results obtained. Theories, discussions, and arguments are all unavailing unless results show their truth. In medicine, however, it is sometimes difficult to demonstrate results until after the lapse of time, as was well illustrated by the preposterous claims first put forth for "606," which is gradually attaining about its correct status. The same is true in regard to the surgery of carcinoma, where operations which formerly were attended with very bad results have gradually been improved, so that, while the late Dr. Agnew of Philadelphia once said, toward the close of his life, that he did not know if he had ever been justified in any operation he had performed on

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this disease, the best surgeons are now claiming notable successes, with relatively good percentages of recovery: but here again, time comes in, and while the three-year limit of freedom from recurrence is valuable, it is still said that fully 90 per cent. of those who have once been affected with true cancer, die of the disease, and its general mortality is still increasing in spite of all surgery. Dr. John B. Murphy of Chicago, has also repeatedly expressed himself most pessimistically in regard to the ultimate results of the surgical treatment of carcinoma, especially in those patients who are fat, and with lax tissue, that is, exhibiting evidences of imperfect metabolism.

Dietetic and medical treatment of cancer, in the fullest sense, have never yet been given a fair and fully intelligent trial on a scale large enough to produce general conviction in regard to their value. Many cases have occurred here and there which

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have recovered spontaneously, that is, without surgical interference, and often really to the surprise of the medical attendant: this of itself shows that under certain circumstances something may occur in the system which causes the malignant process to cease, and the cells to return to a normal function. This certainly should be a stimulus to discover what the reason is for this beneficial change, and we believe that it is to be found in what has preceded in these lectures.

When the New York Skin and Cancer Hospital was founded, over thirty years ago, Cancer was included with Diseases of the Skin, in the hope and expectation that in studying the internal causes of the latter light would be thrown on the etiology of carcinoma, which, as we know, is a disease of epithelial elements; and the earlier Report of the Hospital shows but one department, including Cancer with Skin Diseases, with operating surgeons and a gynæ-

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cologist to assist, whenever their services were requested by the physicians in charge. Before long, however, for reasons which cannot now be given, and against the judgment of the present speaker, the plan was changed, and a separate cancer department was erected under the exclusive charge of surgeons. A Prize Fund was also early established, which has been slowly increasing, for the discovery of a cure for cancer by other than surgical removal.

For thirty or forty years I have held very much the same views regarding cancer which have been presented in these lectures, and have acted on them more or less strongly, and in later years very stringently, and have rarely seen cause for regret. For one reason or another I have also had many hundreds of patients, with various affections, on a vegetarian diet over varying periods: in a number of cases I have records extending over many years,

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in which I have observed the patients from time to time (one psoriasis patient having continued it for over twenty years) and with only the best results, so I trust that you will accept and test the validity of my statements.

In looking over my case histories in private practice I find that I have more or less complete records of 96 cases of carcinoma, mostly of the breast (two of them having Paget's disease), 29 of sarcoma in various situations, and 619 of epithelioma of different degrees of severity, of which four others had Paget's disease; a total of 744 patients with these forms of neoplasm. Some of these patients were seen in consultation, others only once or for a brief period, but many of them were under care and observation for a greater or less length of time. During the past year I have also treated on the plan here indicated a number of cases of recurrent carcinoma in the New York Skin and Cancer Hospital,

which have been submitted to very careful laboratory study, some of whom have exhibited decidedly favorable results: some of these will be referred to later, but they are too recent to afford positive data, and I prefer to confine my consideration mainly to private patients, in regard to whom more reliable evidence can be obtained.

I will not weary you with any full analysis of these cases, nor will I consider any of them except those of carcinoma: for while I believe that sarcoma is of the same nature and origin, save that it relates to connective tissue cells, but few of the cases were submitted long enough to a vigorous treatment calculated to yield efficient results. I may say in regard to the cases of epithelioma, mainly of the skin, that some of the more severe ones showed very markedly the benefit of dietetic and medicinal measures of the character now being considered, and that it is my custom to treat such in the same manner; for reason

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would seem to show that the same causes would produce abnormal proliferation of epithelial elements on the cutaneous surface as well as in other parts of the body.

In regard to the cases of carcinoma there were 6 in males and 90 in females; of these the right breast was affected 42 times, the left breast 45 times, and both breasts 3 times. In 28 cases the patients were operated on surgically before adequate treatment had been employed; many of these were earlier cases, or those seen in consultation. In seven instances it seemed wise to have an operation, after a more or less faithful trial of medical treatment. There were no cases of cancer of internal organs, except metastases, as these would not naturally come to me. The average age of the breast cases was 51.8 years.

In the large majority of cases, except those who had been surgically operated on, the diagnosis was clinical only, as it is an accepted fact that it is very unwise to make

a biopsy, even before surgical removal, and manifestly improper where this is not undertaken; but in most of the cases there was the independent diagnosis of one or more other medical men, and many of the patients came to me on account of threatened operations by prominent surgeons, and even after these had been arranged for; in 27 cases there had been previous surgical removal with recurrence, and in several cases more than one operation, with again recurrence.

The exact duration of cancer before coming under observation is always an uncertain item, for undoubtedly a tumor starts in an individual cell or group of cells, and has probably been forming some time before being noticed by the patient or attracting attention in any way. The average stated duration of the disease was 1.6 years before coming under observation.

Surgeons are fully agreed that the earlier a tumor is recognized and removed

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the better the prospect of ultimate success, and this is eminently true in regard to remittal measures other than surgical: for as we have seen, after a cancerous process is well under way its cells secrete a something which hastens the general depravement of the system, and quickens the growth of the neoplasm, and naturally greatly increases the difficulty of overcoming the dyscrasia.

Thus in the effort to get at and remove all possible or prospective cancerous lesions, many innocent or non-malignant adenomata, etc., are operated on, which are afterwards found to be such microscopically. In the same way it is quite possible that some of the small breast tumors which have disappeared under the dietetic and medical measures here described were still non-malignant, and should not be grouped with carcinoma.

It is fair to say, therefore, that among my notes are also 22 cases of breast tumors

which have been excluded from the carcinoma list, and entered with other titles, such as abscess, cyst, chronic mastitis, adenoma, etc. But I claim that even if some of these also disappear under the measures we are considering, as they frequently have done, it is a better and more desirable result than if they had been removed by the knife: for in the former case the cause which induced the unnatural growth has been overcome, whereas by surgery only the obnoxious mass has been dislodged, and perhaps with it adjoining tissue and glands, but nothing has been accomplished toward checking the cause producing the offending lesion. But the very fact of the frequent recurrences after operation, in the neighboring skin or elsewhere, demonstrates the fact that surgery is but an attempt to rectify past errors, which might or might not have been prevented by careful medical foresight and action; we must, however, be thankful for the meas-

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ure of success which has followed the noble efforts of our distinguished surgeons, only it is to be hoped that hereafter other measures will also be more commonly adopted, looking towards a prevention of the recurrence of carcinoma after operation.

Not to detain you too long with these clinical considerations, I would like to give the details of a few of the cases which have been most striking.

Mrs. B. E. C., aged 44, was first seen on account of trouble in the right breast, on September 19, 1892. She then had a flat tumor in the outer lower segment, an inch and a half in diameter, rather sharply defined, and tender on pressure at the sides, which she had noticed about two months. Not satisfied with the diagnosis of cancer, and hesitating at the thought of medical treatment, she consulted a well-known, prominent surgeon, who pronounced the tumor as undoubtedly cancer, and urged its instant removal; this I did not know until

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she informed me of it some time later, after the tumor had entirely disappeared under treatment. I saw her at frequent intervals for six months, and the breast became entirely normal. Four years later she was again seen in regard to the menopause, which she was undergoing, and the breast was found still to be normal: she was maintaining her diet. Nearly three years later I learned from her husband that she was in perfect health, with absolutely no breast trouble, and for eight years later, while he himself was under occasional treatment, I learned repeatedly that she remained still perfectly well, over sixteen years after beginning treatment, with no recurrence of the breast tumor.

Miss B. M. L., aged 45, was first seen on January 4, 1894, with a tumor in the left breast, which had been diagnosed as cancer by three medical men, and one of them, a surgeon of prominence, had arranged for immediate surgical removal the next day.

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The mass was situated in the upper and outer quadrant, well defined, not painful on moderate handling, but subsequently she experienced pain in it. Two months later the lump was recorded as less distinct and flatter, and within eleven months it had entirely disappeared. A month or two later she had some pain in the breast, in connection with the menstrual disturbance accompanying the menopause, but no trace of the tumor. On Nov. 8th, 1905, she called, bringing a relative for treatment, and the breast was found perfectly normal, and again five years later she called, with another trouble, the breast still remaining perfectly well, also sixteen years after first coming under treatment.

Miss J. M. A., aged 45, was first seen October 12th, 1905, with a tumor of the left breast, above, toward the median line, near the nipple, not painful on handling, though there was some pain afterwards, and it had been awaking her with pain at night

for some months, with also a numb, shooting pain in the daytime. She had seen a number of medical men always with the diagnosis of cancer, but she had declined operation. Two months later, Dec. 15th, 1905, it was recorded that there was very little to be felt in the breast, and there was no pain at any time, and on January 5th, 1906, both breasts were the same on palpation, with no sign of the former tumor. For one reason or another she has been seen from time to time, almost up to the present date, and remains perfectly free from her former trouble, a period of over nine years. She has been a most faithful patient, adhering strictly to diet and more or less medication, except when on certain occasions I have let up on the same; and all the time from the first she has been under great strain of work, mental and physical, as a city missionary.

Another very similar case was in the person of Miss G. M., aged 44, who has

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been under constant observation and treatment off and on for various troubles from November 13, 1905, until yesterday. Fifteen years previous to her first visit she had fallen and struck the left breast which was bruised, but the effects wore off in some months, and there was no sensation in the breast for some years. Then 7 or 8 years later she began to have pain in the breast, aggravated before and during menstruation, which had continued until her visit, and for the last months she had been kept from her work in school, the pain being severe in the breast, and more recently in the axilla: in July there had been bleeding from the nipple. She had seen a number of medical men, with the diagnosis of cancer, and one surgeon of prominence in one of the large hospitals had strongly pressed for an immediate operation.

When first seen there was a tumor between two and three inches in diameter, in the upper, inner segment of the left breast,

hard, sharply defined, and more or less nodular on the surface: there were some enlarged glands in the left axilla. She had long been constipated and was passing about 60 per cent. of the amount of urinary solids proper for her weight. Under very active treatment it was recorded four weeks later that there had been hardly any sensations in the breast during the previous week, that the tumor had diminished materially in size, with only moderate hardness, and that she was now out doors every day, and feeling much better. One month later it was recorded that the breast was very well and on examination was almost the same as the other, there being some general caking in both: she had had no pain for some time. One month or so later she was again at her duties as a public school teacher, which she has continued at since, with rare exceptions, when some temporary ailment prevented: the lump in the breast did not wholly disappear for a

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month or two later, but on April 7th it was recorded that the left breast was the same as the other, and no glands could be felt in the axilla.

From that time to the present she has had a variety of troubles, rheumatic and other, and it has been difficult to keep up a proper action of the bowels and kidneys; but in spite of strenuous and often exhausting work as a New York City public school teacher, she has had no return of the breast trouble, now for over nine years. A sister, aged 60, has just died with cancer of the stomach in a distant country town.

I could multiply these histories but do not want to tire you, though I do want to mention one more patient, to show what can be done in the case of recurrent carcinoma, after operation, of which I have had a number of cases with varying results, according to the duration and severity of the disease.

Miss H. M., aged 61, came to me June

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21st, 1913, with the following history: About two years previously a lump appeared in the outer, lower segment of the left breast, which was removed in August, 1911; this healed soon, leaving a good axillary scar, and there was no thought of trouble until two months before her visit, when a small red spot appeared near the edge of the sternum. This enlarged and hardened and others appeared around the scar, until, when seen there were a dozen red nodules up to half an inch in diameter, near the center of the former site of the breast, with others, not red, above; with the tense skin and rapidly developing, multiple nodules here and there, further surgical operative procedure was out of the question.

Since that date she has been under strict vegetarian diet and medication, including thyroid from time to time, with repeated application of X-rays, and under all these measures together many of the nodules have disappeared; although some

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new ones have formed, several of which have been removed under local anæsthesia, the wounds healing kindly. As she resides some distance from the city she has not been seen since October 7th, 1914, she wishing to take the X-rays nearer home, but she was earnestly charged to continue also the dietetic and other treatment. In this instance the patient has lived comfortably and without pain for almost sixteen months after coming under treatment, and, although she has lost some flesh, the active cancerous process, which would otherwise have carried her off long ago, has been in a measure checked. What will be the further history of the case one cannot tell, for it is quite possible that being away from my care she may neglect dietetic and medicinal treatment, trusting only to the X-rays, which, of course, cannot influence the real nature and course of the disease.

Reference was made to certain cases of

recurrent cancer in the Hospital which had been under active medical treatment during the past year, with careful laboratory studies, but it is naturally too early to report anything very definite in regard to them, especially as most of them were desperate cases, which had advanced far beyond any possible operative relief. Moreover several of them remained but a short time under treatment, as it is very difficult to convince this class of patients that any possible benefit can accrue from anything but an operation, and this being impossible they often give up and leave, preferring to die at home; moreover the dietary restraint seems also very irksome and useless to them and their friends. In one particular patient, however, there was such a remarkable improvement that it is worth reporting to you.

Mrs. C. M. was first seen February 12th, 1914. Nineteen years previously she had an abscess of the right breast, which

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healed and left a tumor the size of a pigeon's egg in the inner, upper quadrant of the breast: this remained quiescent until it began to enlarge, eleven months before it was removed at the New York Skin and Cancer Hospital, November 14th, 1912. The tumor was then about the size of a hen's egg, with an area of skin the size of a quarter, attached to it: a second tumor was felt just below the nipple, which was not retracted, and the axillary glands were involved: there were no signs of metastases in the abdomen. A complete operation was then performed, with dissection of the glands in the axillary and supra-clavicular regions, and she was discharged January 16th, 1913.

On February 9th, 1914, she returned to the Hospital and was placed under medical treatment, with vegetarian diet. There was then an ulceration along the line of incision, from the second to the fourth rib, with many nodules around it, averaging a

third of an inch in diameter, raised and reddened. The liver extended two inches below the edge of the ribs, with a hard and nodular margin; the right arm was enormously swollen and helpless. When she left the Hospital, June 20th, 1914, the ulcer had entirely closed, many of the cutaneous nodules had entirely disappeared, the arm had returned to normal size, like the other, by measurement, and the liver had retracted to a trifle below the margin of the ribs, with hardly any nodules to be felt. The treatment had included twenty-five X-ray exposures, from 8 to 10 minutes each, about twice a week, over three areas each time.

While in the Hospital careful laboratory investigations were made, according to a definite schedule. The *blood*, studied weekly, maintained a hæmoglobin of 80+ for over two months, then fell a little, and again rose. The erythrocytes were 3,262,000 on entering, and rose within two

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1908-10. Then fell a little and rose again to almost 400,000: the haemoglobin was 1000 on entering, and fell to 500 just before leaving, the poly-nuclears per cent. on admission, fell to 40 per cent. and again rose a little, and the proportion of the other forms remained about normal. The urine, volumetrically analyzed every three days, was kept a little below the normal acidity, and the specific gravity a little low, with a free daily amount of excretion, largely by Bethesda water; in spite of the vegetarian diet the urine excretion was not much below normal, and sometimes above, the chlorides were diminished, owing to the rather small amount of food taken, the phosphates varied a little above and below normal, there was never any indican, and the sulphates averaged a little below normal. The saliva, tasted before and after each meal, was not neutral, but became neutral and alkaline and acid. The weight, taken weekly,

fell a little from the first, but maintained a good level, and rose a little before she left the Hospital.

This was a very difficult patient to manage, as she was a very ignorant Polish woman, who often rebelled at the diet, and wearied of the routine and restrictions imposed; she left the Hospital June 20th, 1914, against my wish, but with as great a change in her physical condition and disease as could be imagined, after about four months and a half treatment, carried out under disadvantageous circumstances.

One other case, seen recently, where the disease was recurrent after three operations, deserves mention, although it will be some time before any decisive result can be reported.

Mrs. W. C., aged 45, was first seen September 17th, 1914. Nearly four years previously she had noticed a lump in the left breast which was removed on January 6th, 1911, but it soon regrew, and a complete

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operation was performed at the New York Skin and Cancer Hospital, May 30th, 1911. Two years later there was some return, and she was again operated on at the Hospital, May 30th, 1914. About two months before her first visit, September 17th, 1914, a swelling of the sternum was noticed, and soon another above it, both of which increased pretty rapidly to the time of her visit. When seen there was a hard mass in the scar over the sternum, about an inch and a half long, raised a quarter of an inch or so, reddened and immovable: an inch or so above it was another, smaller one, not reddened: they were not particularly painful on moderate handling, but painful when at hard housework. When last seen, December 7th, both lumps had subsided fully one-half, there was no pain at any time, and her general condition had improved immensely, she feeling better than she did four or five years ago, that is, before the beginning of the cancerous de-

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velopment: she has been working all the time, unusually hard, as janitor of four buildings and also going out scrubbing and washing. She weighed $157\frac{1}{4}$ pounds when first seen, then ran down to 154, but is again gaining, being $155\frac{1}{2}$ at her last visit. The outcome of this case it is, of course, impossible to conjecture, for one can seldom be sure that patients will be absolutely faithful to treatment, for a long enough time, but certainly the change in the woman and in the lesions in this two months and a half has been remarkable, compared with the increasing development of the disease in the two months previous.

I must mention one more case, which, although fatal, exhibited some of the good results of careful medical treatment even when a primary case had advanced far beyond the possible aid of surgery.

Mrs. M. B. J., aged 68, a private patient, was first seen on February 17th, 1914. Two years previously she noticed a lump

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in the upper part of the right breast, after great and repeated mental distress from the death of a number of very near relatives, and a sister's mental derangement; the great nervous strain had been attended with various bilious attacks and nervous indigestion. The mass increased steadily in size and was kept concealed even from her family, until the day before she called, when her family physician who was consulted saw that it was far beyond the possible hope from any operation, in which view a surgeon concurred.

When seen the whole breast was involved, was double the size of the other, hard, immovable, and with an adherent crust over an ulcerating surface on its lower half, several inches in diameter, from beneath which was a moderate discharge: the axillary glands were enormously enlarged, as also the supra-clavicular, and she was strongly cachectic. She was placed on an absolute vegetarian

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diet, with no coffee or tea, and appropriate medical treatment, and the breast kept painted with fifty per cent. ichthyol and water, care being taken not to disturb the adherent crust. In a very short time the discharge ceased, and the protective crust adhered until her death from exhaustion, with pulmonary œdema, on September 9th, 1914. On August 15th it was recorded that the breast had done very well, that it was soft and movable, and not larger than the other breast, with no discharge, and no pain since a short time after beginning treatment: the axillary glands had diminished three-quarters in size, and the supra-clavicular glands were also very much smaller.

And now, gentlemen, my task is done. I have tried to let you see cancer through my spectacles, as I have seen it for very many years past, and to share with me my optimism in regard to the prophylaxis and cure of cancer, if only there can be sufficient enlightenment in the profession and

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public: and I must tell you that in collating and preparing the material to support my long held views I have expended very much more time and labor in study, for some months past, than I could have believed possible. But as the subject developed, and as I discovered more and more support for my thesis, there was a fascination about the work which I could not resist; and if I have tried you with the many details of proof presented I beg that you will pardon me: for I wanted to present the subject so strongly that my hearers, at least, would accept the propositions I have developed, and believe what I have said in regard to my own experience with the terrible disease under consideration, and act upon both, and thus aid some sufferers with cancer.

From my recent article on "The Relation of Diet to Cancer" many medical journals have quoted me as ascribing the disease wholly to the use of meat, but you

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who have heard these lectures now know that animal proteids are only one of the contributing causes.

I have tried to make it plain that metabolic errors, inducing a vitiated blood stream, are the basic cause of the aberrant action in the cellular elements of the body which may ultimately lead to malignant disease; and I have tried to show that there are many elements connected with modern so-called civilization which conspire to effect this end. I have quoted many who were well acquainted with cancer, who believed that luxurious living, which includes much animal food, coffee and tea, and alcohol, with indolence or want of sufficient muscular activity to burn up the waste products, and the persistent neglect of hygienic laws, should be placed first among the causes of cancer: but I have also mentioned that the refining and preparation and cooking of food prevented a proper supply of the mineral and other elements

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of nutrition, and also that nervous influences could so disturb the action of the organs of the body that they could not perform their functions perfectly in the elaboration of nutritive material, etc.

But I cannot go over again all the matter already given in these lectures, and only mention these to remind you that there is no one single cause of cancer, and consequently that its prophylaxis and cure can never be found in any one single remedy; hence I can never believe in the sole use of thyroid, much less in the idea that sero-therapy can overcome a disease dependent upon the continued operation of so many causes; and still less can I believe that the mere cutting out of an already diseased portion of the body is the proper and only means of overcoming such a malady as cancer.

I have acknowledged that local irritation of many kinds may be the proximate cause for the development of a malignant tumor

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in any particular locality, as Ewing has so clearly shown in his excellent resumé on pre-cancerous lesions; but I have also contended that we should withal look into and overcome the cause; why, when once started by local injury the cells should pursue such a progressive, aggressive, and invasive course; and this is found, I believe, in the disturbed character of the fluids which provide them with nourishment for their abnormal growth.

From this study of cancer in regard to its nature, frequency, geographical distribution, metabolism, dietetic relations, medical treatment, and clinical considerations, what conclusions can be drawn? Have we solved the problem of cancer? Far be it from us to make any such claim. Scientific research and study must still go on in the laboratory, but clinical research and study, with laboratory work, on the human subject, which have not been hitherto sufficiently cultivated, should be

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pushed, so that by a mass of carefully recorded observations the truth or falsity of what has been here quoted and said may be refuted or confirmed.

From the enormous work which has been done on cancer with the microscope and the test tube, it would seem sometimes that research workers have become somewhat myopic, and not farsighted enough to recognize the true value of statistical studies and clinical observations. In these lectures we have attempted to make a brief synthetic study of some of the work which has been done in connection with cancer, and from this we believe that certain conclusions can be drawn; if these are correct and followed, it is hoped that much more can be accomplished in regard to the prophylaxis and cure of this more than threatening, fatal malady. In order that you may hold clearly the points which have been made I want to give you a synopsis or conclusions of them, as they have been

brought out in this and preceding lectures.

1. Cancer is but a deviation from the normal life and action of certain of the ordinary cells of the body, which, for some as yet unexplained reason, take on an abnormal or morbid action; with this there is a continued tendency to a malignancy which invades contiguous tissue, and in the end tends to destroy life.

2. There is some reason to believe that this action first takes place in what are known as "embryonic rests," or pre-natal, wrongly placed tissue elements, which, however, are now shown to exist in every one, in many localities: but the reason why they take on this malignant action, and form cancer, has not been satisfactorily explained.

3. Cancer is *not* wholly due to traumatic causes, although those play a not inconsiderable part in its occurrence in certain localities and cases.

4. It is pretty conclusively decided that

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cancer is *not* caused by a micro-organism, or parasite.

5. It is also known clinically and experimentally that it is *not* contagious.

6. *Nor* is it hereditary in any appreciable degree.

7. Occupation has *not* any very great influence on the occurrence of cancer, although it is more frequent in some pursuits than in others.

8. Cancer is *not* altogether a disease of older age, although its incidence is greatly increased with advancing years.

9. It does *not* especially belong to or affect any particular sex, race, or class of persons.

10. It is *not* confined to any location or section of the earth, but has been observed in all countries and climates.

11. No single cause of cancer has yet been demonstrated, nor is it likely that this will ever be the case, as the experimental and other investigations have cov-

ered almost every possible line of research, with only *negative* results.

12. The exclusion of almost every other possible cause of cancer, as well as its pathological history, leads to deranged metabolism as the only remaining possible etiological element; this acts by inducing changes in nutrition, which latter depends on diet and the proper action of the secretory and excretory organs, which, still further, may be affected by nervous influences.

13. While the bio-chemistry of cancer throws little light on its true nature, enough is known to show that the morbid changes in the cells are largely associated with deranged metabolism.

14. The blood, in advancing cancer, manifests changes which indicate vital alteration in the action of the organs which form blood and control the nutrition of the body and its cells.

15. Clinical and experimental evidence

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demonstrate that the secretions and excretions of the body exhibit departures from normal, which, while not pathognomonic of cancer, indicate metabolic disturbances involving the nutrition of the cellular elements, which disturbances are of importance.

16. The evidence seems certain that the cancer mass itself, when fully developed, secretes a poison which tends to augment its own growth and hastens the lethal progress of the disease.

17. Cancer mortality is undoubtedly on the increase in every portion on the globe.

18. This increase seems to vary inversely with the decline of tuberculosis, in many localities.

19. The incidence of cancer seems to follow closely along the lines of modern civilization.

20. This extension of cancer seems to depend largely upon the altered conditions of life, particularly along the lines of self-

indulgence in eating and drinking, and indolence.

21. The augmentation in the consumption of meat, coffee and alcoholic beverages appears to be coincident with a very great, and proportionately greater, augmentation of the mortality from cancer.

22. The nerve strain of modern life seems to be an element of importance, both through disturbance of metabolism, and by direct action on morbidly deranged cells.

23. No single remedy for cancer will probably ever be discovered, since it is conceded that there is no single cause for the disease.

24. Surgery has improved materially the statistics relating to the mortality of operative cases; but the total achievements along this line are insignificant compared with the general ultimate mortality of over 90 per cent. of those once afflicted with cancer.

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25. Surgery has had and may long have its function to perform in removing the products of the disease, more or less efficiently, curing some and prolonging the life of others, but it can never hope to lessen greatly the morbidity of cancer.

26. The X-ray and radium, as also caustics, are in the same position as surgery, and can do relatively little more than cause to disappear lesions which have developed from causes which they cannot reach.

27. With all these means the measure of success, aside from the technical skill of the operator, largely depends on the time or period of development of the malignant tumor before treatment; the earlier the removal is undertaken, other things being equal, the greater the possibilities of success.

28. The same is true in regard to the treatment of the disease by means other than those mentioned: the earlier the morbid process leading to tumor formation is

attacked by dietetic, hygienic, and medicinal measures, the greater the promise and expectation of success, present and permanent.

29. The prevention of cancer, therefore, or the checking of its increasing occurrence, depends largely upon the early enough adoption of such measures as will limit the agencies which induce a derangement of the body juices which tend to bad nutrition and derangement of the body cells.

30. The simple life, with the avoidance of the dietetic and other causes which have been found to induce cancer in nations and individuals, promises the best hope for the arrest of the rapidly increasing development of cancer throughout the world.

31. It is more than possible, however, that the long continued operation of many baneful causes has produced such a degeneration of tissue in the human race, that it will take a generation or more of proper

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living to make the beneficial impression on the general incidence of cancer which is so longed for.

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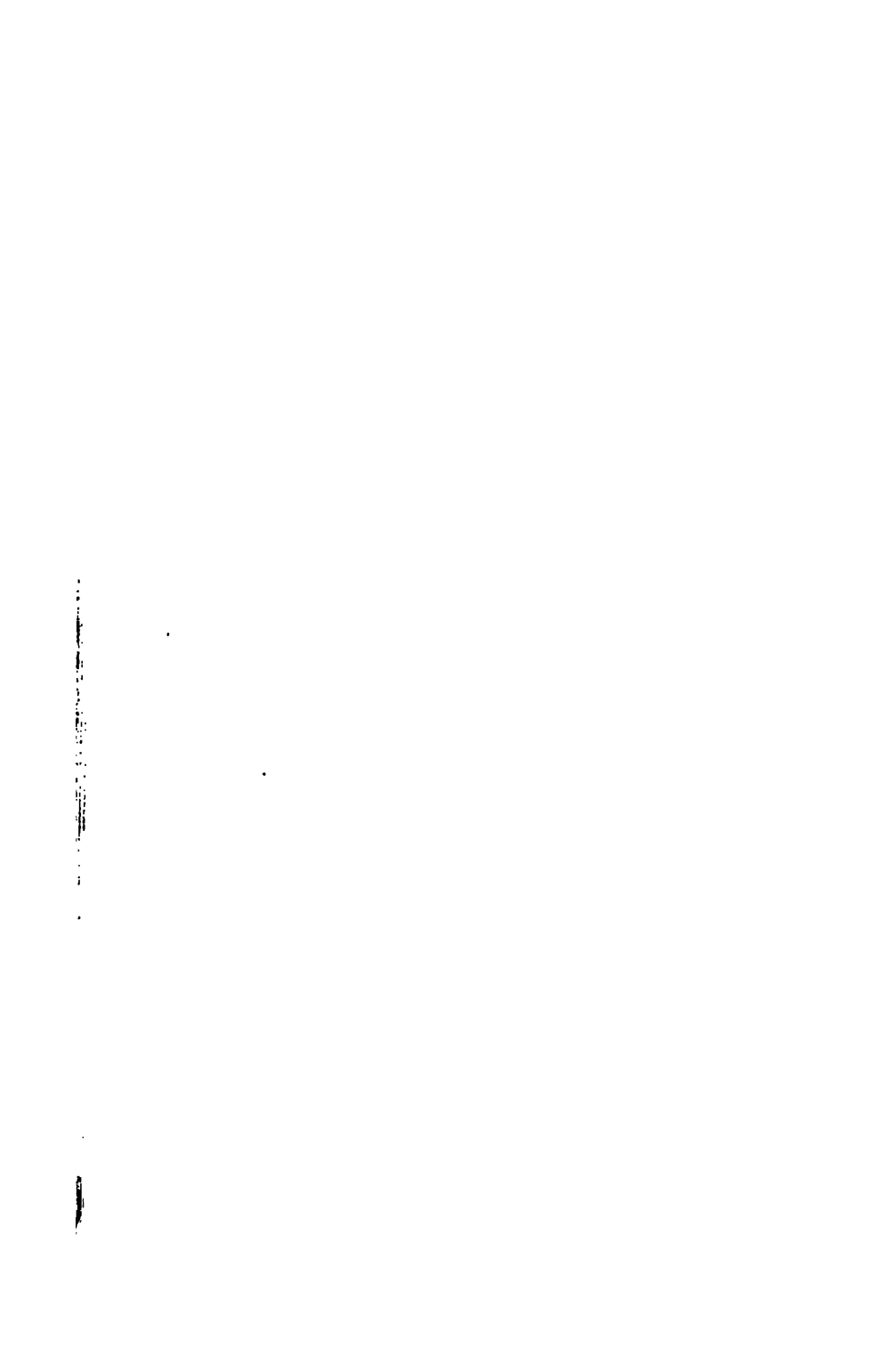
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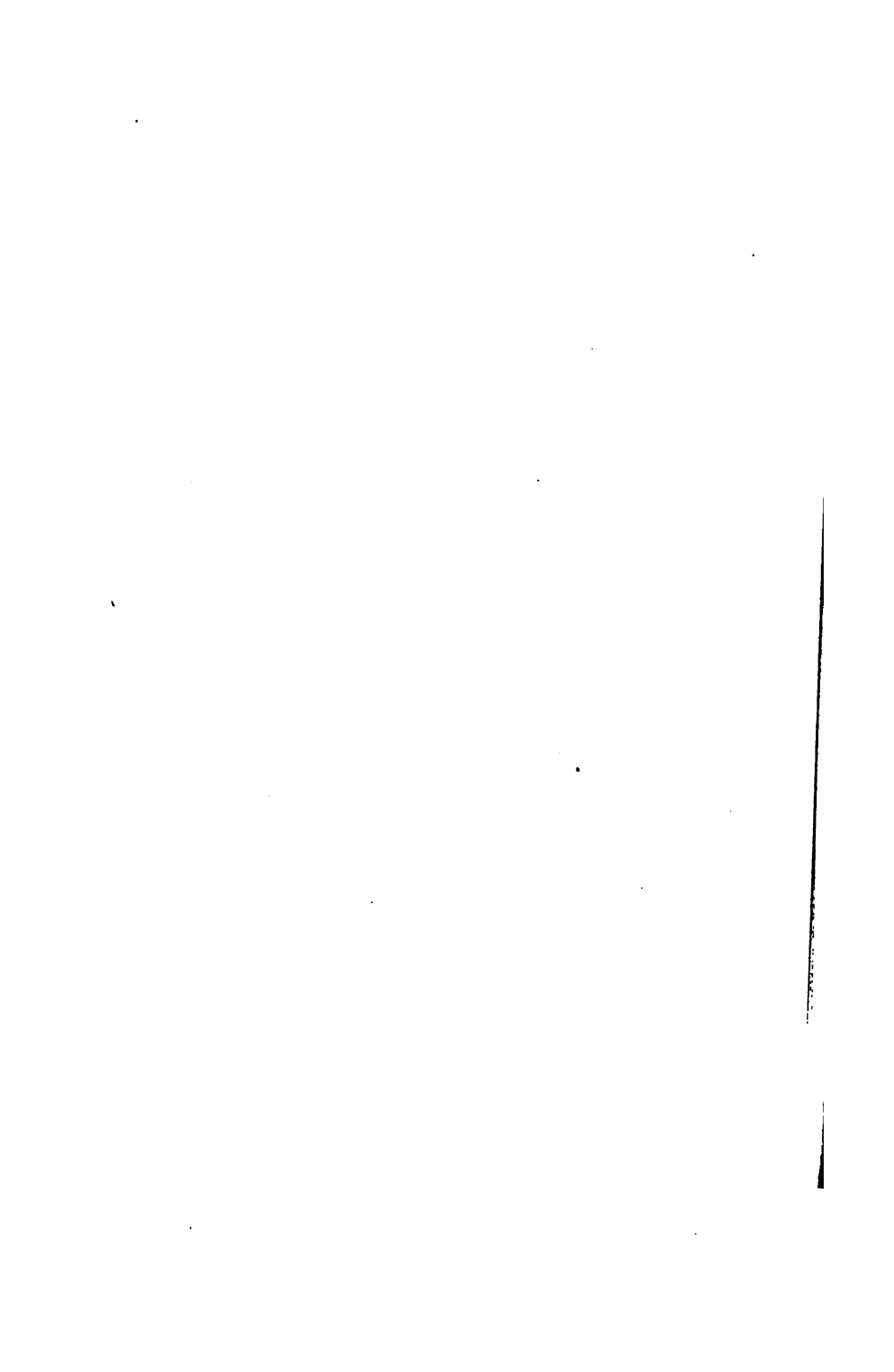
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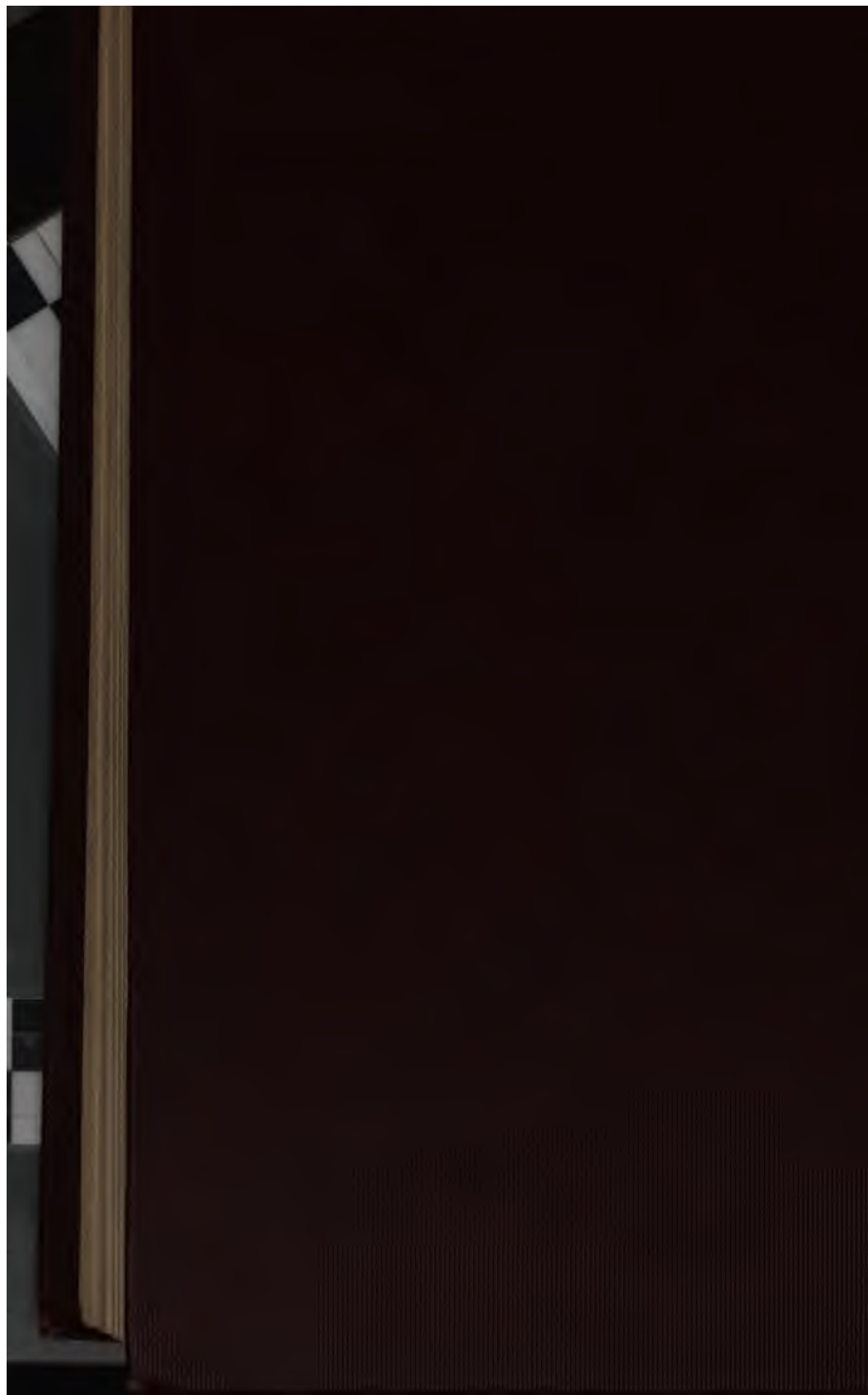
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THE ELEMENTS OF CLINICAL DIAGNOSIS



THE METHOD OF DIAGNOSTIC EXAMINATION

THE tasks of practical medicine are prophylactic and curative. A systematic treatment of disease rests upon the recognition of its forms and manifestations. The science involved in the recognition of disease is called *diagnosis*.

A complete diagnosis includes: (1) The naming of the disease, *i.e.*, its classification or place in some particular group of diseases; (2) the recognition of the stage of the disease, peculiarities or complications; (3) the appreciation and estimation of dangers existing or liable to arise.

A diagnosis is reached by the examination of the patient. This consists in the obtaining of the history (anamnesis) and in the objective examination (*status præsens*). It is always advisable to follow the order of a systematic scheme in order to avoid mistakes of omission.

The following scheme of examination has long been used in the first medical clinic of Berlin:—

Name, age, civil condition. Date of examination.

Anamnesis:

1. Hereditary relations.
2. Diseases of childhood, menstruation.
3. General conditions of life, occupation.
4. Previous diseases, puerperia.
5. Present illness, its prodromata and suspected cause.

2 THE METHOD OF DIAGNOSTIC EXAMINATION

6. The first phenomena of the disease. (Chills and fever? Subjective complaints, functional disturbances.)
7. Course of the disease to date.
8. How long did it increase in severity? Has it been better or worse?
9. Previous treatment.
10. Complications: data of patient as to the principal functions; e.g., sleep, appetite, cough, expectoration, urine, etc., condition of nutrition and strength, appearance.

Status præsens.

A. General Examination.

- I. *Constitution* (stature, osseous system, muscles, fat).
- II. *Position* (active or passive, recumbent position, etc.).
- III. *Face*.
 1. Color (cheeks, lips, conjunctivæ).
 2. Nutrition, congestion.
 3. Expression.
 4. General appearance.
- IV. *Skin*.
 1. Color (p. 7).
 2. Eruptions, œdema, scars, decubitus.
 3. Moist or dry.
 4. Temperature and its distribution.
- V. *Pulse*.
 1. Frequency, rhythm.
 2. Condition of the arteries (straight, tortuous, sclerosed).
 3. Tension of the arteries.
 4. Pulse-wave.
- VI. *Respiratory frequency and rhythm*.
- VII. *Striking symptoms*.
- VIII. *Complaints of the patient*.

B. Special Examination.

- I. *Nervous system*.
 1. Sensorium (free or stuporous).
 2. Headache, vertigo.
 3. Sleep.
 4. Tremor.
 5. Delirium, abnormal moodiness.
 6. Disturbances of sensation and motility.
- II. *Digestive system*.
 1. Lips, tongue.

2. Throat.
3. Appetite.
4. Thirst.
5. Vomiting.
6. Stools.
7. Palpation of the abdomen (painful areas? tumors?), the liver and spleen.
8. Percussion of the abdomen (stomach, liver, spleen, tumors).
9. Examination of stomach contents.

III. *Respiratory system.*

1. Rhythm of respiration.
2. Shape of thorax.
3. Respiratory movements (frequency, type, amplitude, unilateral).
4. Cough and expectoration.
5. Percussion.
6. Auscultation.
7. Pectoral fremitus and bronchophony.

IV. *Circulatory apparatus.*

1. Inspection of the cardiac area.
2. Inspection of the large vessels.
3. Palpation of the cardiac impulse.
4. Palpation of the apex beat.
5. Percussion of the heart.
6. Auscultation of the heart.
7. Auscultation of the large vessels.

V. *Urine.*

1. Voluntary, painful evacuation.
2. Amount in 24 hours.
3. Specific gravity.
4. Color, clearness.
5. Reaction.
6. Albumin, sugar.
7. Sediment and its contents.

Note. — The beginner will do well to learn this or some similar scheme well and to adhere to its order in all his examinations. The experienced physician determines the general condition in a few minutes while asking the patient the necessary questions. Through the general condition and the history, attention will be directed diagnostically toward certain organs or systems of organs with which the special examination is then begun. The organ found diseased must be examined with the greatest care; so far as the other organs are concerned, the determination of their main features is sufficient.

CHAPTER I

ANAMNESIS AND GENERAL CONDITION

1. **Anamnesis.**—An exact history of a case is of the greatest importance; for, frequently, the decision of a diagnosis hinges upon it.

Hereditary taint is especially significant in the diagnosis of phthisis and the nervous diseases.

The causes of a present illness may lie in the sequelæ of some previous disease; *e.g.*, scarlatina may be productive of an acute, more rarely of a chronic, nephritis; an articular rheumatism may lead to a valvular endocarditis; repeated attacks of bronchitis and asthma may superinduce emphysema.

Certain occupations may entail certain diseases: painters are subject to lead poisoning, cornetists to emphysema; composers, millers, and stonecutters are liable to acquire phthisis.

Certain noxious substances possess etiological character: alcohol induces cirrhosis of the liver, or a weak heart, or multiple neuritis, or chronic nephritis.

Certain events in the history of a patient are of diagnostic value; *e.g.*, an hæmoptysis (phthisis), hæmatemesis (gastric ulcer), attacks of jaundice (gall-stones).

It is well for the beginner to remember that the taking of the history is often the occasion of the first meeting between patient and physician. To win the confidence of the patient, the questions should always be asked in a friendly and gentle manner, and modified to suit the patient and the occasion.

2. **Condition of nutrition and strength.**—The state of a patient's nutrition is usually easily recognized by a glance at the face (fat or thin, of pale or ruddy color, bright or sunken eyes, lively or depressed expression) and the rest of body (fat, muscular development of the buttocks, of the arms and legs).

The state of the nutrition leads the diagnosis to a particular group of diseases. A poorly nourished condition is indicative of the so-called *cachectic* diseases (phthisis, carcinoma, leucæmia, anæmia, the severer form of diabetes). A patient who maintains a well-nourished condition in spite of long-continued illness is not a victim of cachectic disease. Because of their short duration, the acute febrile diseases do not essentially disturb the nutrition; the sub-acute diseases (typhoid fever, meningitis), on the other hand, lead to marked emaciation.

The nutritive state is of particular importance in the differential diagnosis of the diseases of the lungs and stomach. Phthisis and carcinoma of the stomach are suspected in cachectic individuals; bronchitis, ulcer, and neurotic disturbances of the stomach appear more often in healthy-looking persons.

3. Constitution and habitus. — Through the repeated observation of the sick, the physician secures certain impressions of single forms of disease, by means of which, on seeing similar cases, he at once suspects the nature of the illness. These impressions are a composite of the nutrition, complexion, appearance, position, speech, etc. The judgment as to the habitus is of unquestionable worth, but must not preclude a careful examination.

Habitus phthisicus is seen in tubercular subjects. It is characterized by a pale, often spiritual countenance, with a fine skin and circumscribed redness of cheeks; the neck is slender, the thorax weakly acting. The figure is thin and wasted, the hands are small and white.

Habitus apoplecticus is marked by a dark-red, round, stout face, glistening, watery eyes, and short neck. The chest is usually barrel-shaped, and the respiration is of a short, wheezy character, indicative of emphysema. The body is abnormally fat. If the patient is alcoholic, he has a tendency to apoplectic insults.

With the *habitus neurasthenicus*, the patient is usually well nourished and has an expressive face. The eyes are bright, have an unsteady gaze, and are passive as though their possessor were

in suffering. These patients are moody and hypochondriacal, and are often suspicious. Speech often impulsive.

To recognize the habitus associated with a particular disease is largely dependent upon experience, though it is sometimes an intuitive faculty.

4. The position of the patient in bed, which is immediately observed, may modify the general impression as to the character of the disease. If the patient is lying on his back, it must be observed whether he lies with little muscular effort (*active position*), or, yielding to his burdens, he is sunk down in bed, with knees drawn up (*passive position*). The latter position, if maintained, is always a sign of weakness or collapse, and is usually of grave significance.

A patient with disease on one side of the body (*e.g.*, pneumonia, pleurisy, pneumo-thorax) often assumes a *side* position, usually lying on the diseased side. In some gastric affections, the patient lies on the abdomen.

Restlessness or jactitation is a sign of fever (see below), and is often a forerunner of delirium. At the same time, the danger of such conditions as wakefulness and stupor must be regarded.

The *sitting posture* is most frequently observed in the disturbed compensation of cardiac disease, and is a symptom of dyspnoea or orthopnoea.

5. The features, expression, and appearance of the patient give valuable hints in the determination of a diagnosis.

The *facies composita* is the intelligent play of features seen in health. The *facies Hippocratica* or *decomposita* is the fixed, distorted, inanimate appearance of the features in unconsciousness and approaching death.

The physician should learn to estimate a patient's sensorium from his facial expression. In the febrile diseases this is of especial value and importance. Typhoid fever, meningitis, and acute miliary tuberculosis and sepsis, for instance, run their courses with disturbed mental and nervous functions, and the face is then expressionless, dull, and apathetic. The facial expression of patients suffering from

such diseases is very characteristic and may be recognized at the first glance. In other febrile diseases, the face has a turgescient but a simultaneously clear and expressive appearance.

The beginner should learn thoroughly the various types of expression; the study of the physiognomy is of unquestionable diagnostic value, and was highly regarded and constantly practised by the physicians of bygone years, masters of observation. A careful examination must not, of course, be precluded.

6. The complexion and the skin may offer some help in establishing a diagnosis.

The normal pale-red color of the skin, with rosy cheeks and bright-red lips, allows only negative conclusions.

a. *Flushing* of the face, accompanied by heat, turgescence, sweat, and bright, bulging eyes, is usually a sign of fever. Further symptoms of temperature would, of course, be looked for; but one must not forget the fleeting redness due to excitement and modesty.

b. Abnormal *paleness* (*pallor eximius*), chalky or waxy color of the cheeks and body, pallor of the lips and conjunctivæ is a sign of anæmia. The anæmia may be *primary* (essential anæmia) or *secondary*. It is primary when dependent upon disease of the blood, as proven by an examination of the blood (Chap. XI.). It is secondary when it follows or is coincident upon severe systemic disease (phthisis, carcinoma, amyloid degeneration, etc.). The sudden appearance of an anæmia which has a continued duration and which is preceded by collapse, speaks in favor of an internal hæmorrhage: in the stomach, in the intestine, or in the Fallopian tube.

c. *Jaundice* (*icterus*) is a sign of the deposit of bile-pigment in the skin and is usually coincident with some form of *liver* disease. A jaundice occurring in a well-nourished patient usually denotes a catarrhal inflammation of the duodenum and bile-ducts (*icterus simplex*). Jaundice with severe ill-

ness is indicative, as a rule, of grave affections of the liver (*icterus gravis*, Chap. IV.). Independent of liver diseases, the graver form of jaundice may result from toxic absorption, which destroys the red blood cells (*cythæmolysis*), as in acute phosphorus poisoning, or from the products of the acute infectious diseases.

The yellow color of the skin may be induced by the administration of *picric acid*; but the urine will not then respond to the tests for bile-pigments.

The classification into *icterus simplex* and *icterus gravis* is of great practical value. Of more scientific importance, however, is the division into jaundice with and without *polycholia*. Icterus with *polycholia* is produced by an overflow of bile in consequence of great disturbance of the red blood corpuscles. It is comparatively rare, and leads rapidly either to death or to recovery and is never chronic. The stool is always well colored with this form of jaundice. Jaundice without *polycholia* is caused by a congestion in the bile-ducts and occasions the simple jaundice and the greater number of cases of malignant jaundice. In these cases the stools are usually colorless, the color varying with the degree of occlusion of the bile-ducts.

d. A *bronze-colored* skin is the pathognomonic symptom of Addison's disease, which has its probable lesions in the suprarenal capsules and splanchnic nerves. It is always fatal, and is accompanied by an increasing cachexia. The brown pigmentation of the mucous membrane of the mouth is particularly characteristic.

e. *Cyanosis* is the reddish-blue condition of the tissues seen in certain diseases, most easily recognized in the lips and finger-nails. It depends upon an over-charge of carbon dioxide in the blood. It may be caused by: 1. Slow or imperfect circulation of the blood, and it is, therefore, an important symptom of uncompensated *cardiac* disease. 2. Disturbance in the exchange of gases in the lungs by (a) pulmonary disease, or (b) abnormal distention of the abdomen by tumors, meteorism, or ascites. These conditions

lead to cyanosis, as a rule, only in the advanced stages, whereas at the beginning, or at the height of the disease, there is frequently a vicarious increase in the force of respiration in the unaffected portion of lung. In *pneumonia* the appearance of cyanosis is of grave import. In *acute miliary tuberculosis* there is usually very pronounced cyanosis.

A local cyanosis arises from venous congestion by thrombi or tumors. In the face, it may be the result of freezing.

A combination of cyanosis and pallor (*livor*) is sometimes seen in patients exhausted by heart disease. Organic cardiac disease, with deep congestion, not uncommonly produces cyanosis with jaundice, the latter arising from congestion of, and absorption from, the gall-ducts in the congested liver.

7. **Dyspnœa** (air-hunger, impeded respiration).—The physician must determine at once whether the respiration is normal or not, but should notice especially whether it is free or labored.

It is essential to differentiate between *increased* and *labored* respirations. A simple increase in the number of respirations, above 24 to the minute, is of no particular diagnostic value, and can appear after bodily exertion, as a result of emotional influences, in hysteria or in fever.

Dyspnœa is an indication of the need of oxygen. It is characterized by rapidity and deepening of the respiratory movements, with contractions of the auxiliary muscles of respiration. The patient feels "air-hungry" up to the point of suffocation. True dyspnœa is usually accompanied by cyanosis, a condition pathognomonic of uncompensated heart disease and advanced disease of the lungs. Less commonly they appear together in some abdominal diseases which impede diaphragmatic movement.

Attacks of dyspnœa which pass away more or less rapidly, and are followed by longer or shorter periods of quiet breathing, are called asthmatic attacks.

In *Cheyne-Stokes* respiration, the respiratory movements gradually decrease both in extent and rapidity until they cease altogether, and a condition of apnoea ensues. This is followed by a feeble respiration, succeeded in turn by a somewhat stronger one. The amplitude of the respiratory movements increases and wanes again in a similar manner. This phenomenon is seen especially in uræmia, also in cardiac and brain diseases, and is usually of grave significance. Still, an indication of the Cheyne-Stokes phenomenon is sometimes seen in the sleep of healthy persons.

The description of other changes in respiration is reserved for the chapter devoted to the subject (p. 121).

8. The excessive accumulation of lymph in the subcutaneous connective tissues is known as *œdema* or *dropsy*. The skin above a dropsical part of the body remains pitted after pressure by the finger. *œdema* is such a striking symptom that patients frequently make it their principal complaint; stupid patients may overlook it, however, and it is a good practice to accustom oneself to look for it.

The first signs of *œdema* can be recognized by pressure of the fingers in the neighborhood of the ankles.

The presence or absence of *œdema* determines the path of the diagnosis. If there are no complicating cyanosis and dyspnœa pointing to heart disease, the nutritive condition and the condition of the blood must be considered and the urine must be examined for albumin.

a. Dropsy with cyanosis and dyspnœa is the symptom of uncompensated heart disease: *cardiac dropsy*.

The diseases of the heart interfere in a marked degree with the venous return of the blood. The blood remains too long in the tissues, where it loses its O and takes up more CO₂ than usual. The over-distended veins no longer take up the lymph in the usual quantity, and the latter fluid floods the tissues.

b. Dropsy with albuminuria is the symptom of disturbed function of the kidneys, and is known as *renal dropsy*. Richard Bright (1825), an English physician, first described this combination of symptoms, and it is therefore called "*Bright's disease*."

The dropsy in albuminuria may be explained as follows: normally, the capillaries are impermeable to large quantities of plasma on account of the vital activity of the cells in their walls. This activity is intact only when the cells are well nourished, *i.e.*, when the composition of the blood is normal. The blood becomes deteriorated when the kidneys are diseased; in health, these organs separate all waste products from the blood; in disease of the renal epithelium, metabolic products remain in the blood, the walls of the capillaries become permeable because of poor nutrition, and oedema results. At the same time, all diseases of the renal epithelium lead to the appearance of albumin in the urine.

Another theory attributes renal oedema mainly to an inflammatory process evoked by the action of the same noxious materials upon the walls of the capillaries as induces a similar process in the kidneys.

In oedema due to *great congestion*, there is often albuminuria because of the simultaneous congestion of the renal veins. The diagnosis is turned toward the heart by the accompanying cyanosis and dyspnoea.

In chronic diffuse nephritis with or without exudation and with the atrophic kidney with granular degeneration there is frequently a hypertrophy of the left ventricle of the heart. The heart and the kidney both give symptoms.

c. Edema accompanying *cachexia* can be observed in all conditions of bad nutrition, especially in the cachectic diseases: carcinoma, pernicious anæmia, phthisis, diabetes. It can appear temporarily after excessive exertion and in inanition.

Under these conditions, the oedema can also be explained by the abnormal condition of the blood, the consequent disturbance of the intima, and the permeability of its cells. The impoverishment of the blood arises from disease of the blood (anæmia, severe chlorosis, leucæmia), or from ill-nutrition or from chronic organic disease. The transitory oedema of young people who have excessively exercised may be partly explained by their prolonged upright position.

9. **Exanthemata** (eruptions).—The examination of the skin is incomplete unless eruptions are searched for. These are of particular importance in the febrile diseases in which

they sometimes determine the diagnosis. It is difficult to recognize the various eruptions from description alone. The student should therefore improve every opportunity to observe them.

The eruption of *measles* is scattered and is composed of large macules; that of *scarlatina*, small macules, lying closely together so that it gives an appearance of diffuse redness. In *typhoid fever* there is a *roseola*, sparse over the abdomen, less thickly scattered over the chest, which may vary in size from the head of a pin to a pea. In *typhus*, a profuse roseola. Exanthemata may appear sometimes only several days after the beginning of the fever: the absence of the eruption does not, therefore, exclude the diagnosis of an exanthematous disease.

Exanthemata *without fever*, with little or no general disturbance, may be referred to diseases of the skin often dependent on syphilis.

Occasionally eruptions follow the exhibition of certain *drugs*, such as the measles-like eruption of antipyrin and the acne of potassium iodide. These disappear, naturally, after the cessation of the medication.

Subcutaneous hæmorrhages (ecchymoses, sugillations, or petechiæ when punctate) occur at times: (1) in severe forms of some of the acute infectious diseases, as scarlatina, small-pox, typhoid, typhus, hæmorrhagic measles; (2) in acute articular rheumatism, even in cases not particularly severe, peliosis, and purpura rheumatica; (3) in very severe anæmia or leucæmia; (4) in some diseases of the liver, as acute yellow atrophy, rarely in cirrhosis; (5) in certain diseases due to disturbed metabolism, which lead to great weakness: morbus maculosus Werlhoffi, scurvy. In the last named disease, bleeding from the gums is characteristic.

Petechiæ, which become pustular, are symptomatic of embolism in the skin in severe pyæmia, ulcerative endocarditis, and glanders.

10. Temperature of the skin.—The body temperature can be approximately estimated by the hand placed first on the chest and then gently pushed into the axilla. Elevated body temperature is the principal symptom of fever. If the temperature is above 98.4° F. (37° C.), a differential diagnosis of the febrile diseases must be made. See Chapter II.

The most striking symptom, as a rule, to call attention to a febrile condition, is the flushed face. While the temperature is being taken, the pulse should be felt, and search for an eruption should be made.

If the patient is perspiring or the hand of the physician is cold, estimation of body temperature by the hand is unreliable.

11. **Dryness of the skin and perspiration** can either one be of diagnostic value. Great dryness is present in conditions in which there is a large amount of watery excretion, such as polyuria from any cause, diabetes mellitus, cholera, severe diarrhoea. Perspiration, also, may be of diagnostic importance. In some of the febrile diseases profuse sweating may indicate the approaching crisis. In chronic diseases it may be a sign of weakness, as in the night-sweats of phthisis. It often accompanies collapse and approaching death. Among drugs, the antipyretics of the coal-tar series, especially, occasionally produce profuse sweating. It may appear, however, now and then, in healthy persons, especially in the young, after a large ingestion of fluids.

12. **The pulse.**—By long-established precedent, the examination of the patient is begun by feeling of his pulse. By this procedure one can recognize: *a.* The presence or absence of fever. As a rule, the pulse-rate is quickened (over 90) in the febrile state; the tension is increased while the artery remains soft. *b.* The patient's general condition. A strong, healthy man has a full pulse of good tension; a person weakened by disease has a rapid, small pulse, with little tension. *c.* The presence of particular changes in the heart, or in certain other organs (Chap. VII.).

The pulse is felt by placing the finger (usually the index finger, *never* the thumb) upon the radial artery, a little above the wrist. The hand of the patient should not be raised from the bed. The beginner should accustom himself to count the pulse, watch in hand, for a quarter of a minute, and to determine the minute-rate from this calculation.

The feeling of the pulse is an art which can be learned only by constant and by long practice. Experienced physicians sometimes bring it to an extraordinary degree of perfection. Indeed, some of the most valuable diagnostic hints may be gained by this proced-

ure. The older physicians, who were masters of observation, were accustomed to lay the greatest importance upon this practice.

13. **Striking symptoms.**—As a training in diagnostics, it is a good practice for the physician, after the completed general examination, to ask himself if he has omitted to take note of any striking symptom or symptoms. It is, of course, a matter of experience to observe and appreciate certain signs and symptoms which might easily be overlooked in the general examination, and would come to light only in the systematic examination of each organ.

Any single element in a patient's complex of symptoms may appear as a striking symptom, as, for instance, extreme pallor, dyspnoea and cyanosis or oedema. Certain diseases may produce striking symptoms, the observation of which may be the starting-point of the diagnosis; *e.g.*, *ascites* (fluid in the abdominal cavity), *meteorism* (distention of the abdomen through gas in the intestines), *enlarged glands*, *varicose veins*, *vomiting*, *peculiarities in the urine or sputum*, etc., etc.

Mention must be made of a few striking symptoms which are of more value as to the judgment of the present condition of a patient than for the establishment of a differential diagnosis.

1. A sudden change in the condition of a patient, characterized by a small and frequent pulse, great difficulty in respiration, extreme pallor, coldness of the extremities, nose, and ears, with a rapid decline of the body temperature, is known as *collapse*. It is caused either by an internal hæmorrhage or by sudden heart failure in the crisis, recurrence, or even convalescence of the febrile diseases. In the last stage of typhoid fever and after diphtheria, collapse must be looked for. It sometimes follows a rapid sitting up in bed, too early or too long absence from bed, or over-great exertion at stool; sometimes it appears without any appreciable cause.

2. An accumulation of fluids in the larger air-passages, which gives rise to the well-known *stertor* or *death-rattle*,

is a symptom of approaching death. It can be heard at inspiration and expiration, and at some distance.

3. What is commonly called the *death-struggle* or *agony* is the appearance of the progressive and complete paralysis of all muscular and nervous functions. It is characterized by unconsciousness, the *facies Hippocratica*, and the phenomenon of the death-rattle.

The *certain* signs of death are: cessation of respiration, of the sounds of the heart, of the pulse; absence of all reflexes, particularly that of the cornea.

It is certainly of rare occurrence that a physician is in doubt whether a seeming corpse is really dead. Should such a suspicion arise in the case of a sudden death, certain experiments will readily decide the matter; such as, the exposure and incision of an artery, electric stimulation of the muscles, the insertion of a needle into the heart or the application of down to the lips.

CHAPTER II

THE DIAGNOSIS OF THE ACUTE FEBRILE AND ACUTE INFECTIOUS DISEASES

IN addition to the general considerations, the history, in a febrile case, must embrace: *previous infectious diseases* (typhoid fever, measles, and scarlatina attack an individual but once, as a rule, but pneumonia, erysipelas, and articular rheumatism may occur repeatedly); *direct causes of infection*, such as similar illness in the neighborhood, or exceptional opportunity for infection by food, water, etc.; *predisposing causes* (cold, emotion, trauma, errors of diet); *initial symptoms*: chill, headache, lassitude, sore throat, vomiting, pain in side or back, etc.

The febrile diseases in general can be recognized by the symptom complex of *fever*. This is characterized by a red, flushed face often covered with perspiration; quickened breathing; full, rapid, but soft pulse; pronounced thirst; diminished appetite; diminished quantity of urine of high specific gravity and dark-colored; *increased* body temperature.

The temperature should be taken at once with the *clinical thermometer*. The thermometer should remain in the axilla 10 minutes, in the rectum 5 minutes.

The temperature in the mouth is from 0.2° to 0.3° higher than in the axilla; the temperature taken in the rectum is about 1° F. (0.5°–0.8° C.) higher than that in the mouth. The temperature of a healthy person estimated in the mouth or rectum varies between 98.5° and 99.5° F. (36.9°–37.4° C.). It is lowest in the morning, and tends to rise in the evening from 0.5° to 1° F. or C. The Germans employ the scale of Celsius, the French that of Réaumur, the Americans and English the Fahrenheit scale. The equivalent table is here given:—

$$n^{\circ} \text{C.} = \frac{9}{5} n^{\circ} \text{R.} = \frac{9}{5} n^{\circ} + 32^{\circ} \text{F.}$$

C.		R.		F.
36°	=	28.5°	=	96.8°
37°	=	29.6°	=	98.6°
38°	=	30.4°	=	100.4°
39°	=	31.2°	=	102.2°
40°	=	32°	=	104°
41°	=	32.8°	=	105.8°

The so-called *self-registering thermometers* may be recommended. After the taking of the temperature, a little metal rod lying above the mercury shows the degree of fever until shaken down.

The *minute thermometers* are handy for general use. Because of their smallness and of a peculiar amalgam of mercury, they give the accurate axillary temperature in two minutes.

The temperature of healthy persons in the axilla is from 36.5° to 37.5° C. It is lowest in the morning, in the evening from 0.5° to 1.0° higher. Slight elevations of temperature appear *transitorily* after a heavy meal (digestive fever), great exertions, continued exposure to the sun (insolation), warm baths. Continued elevation of temperature is a sign of *fever*. Temperature below 36° C. is called *collapse* temperature; 36° to 37° C. normal; 37.5° to 38.0° C. *subfebrile* temperature; 38° to 38.5° C. *light* fever; 38.5° to 39.5° C. (evenings), *moderate* fever; 39.5° to 40.5° C., *high* fever; above 41.5° C., *hyperpyretic* temperatures.

When fever exists, the temperature varies during the day; in the morning there is a moderate decrease (*remission*), in the evening an increase (*exacerbation*). If the exacerbation comes in the morning, the remission in the evening, an *inverted type* of fever is present (most frequently in phthisis).

Chills: If the bodily temperature rises very suddenly while the radiation of heat is diminished by contraction of the cutaneous vessels, the patient has the sensation of intense cold which manifests itself in involuntary shivering, chattering of the teeth, shaking of the entire body. A chill appears: 1. in a single attack at the beginning of the acute infectious diseases (pneumonia, erysipelas, scarlatina, etc.); 2. in repeated attacks: (a) at regular intervals: in malaria (which may be suppressed by quinine), (b) at irregular intervals (uninfluenced by quinine), in the presence of deep abscesses and pyæmia, more rarely tuberculosis and endocarditis. — Chills in the course of a typhoid fever may indicate a relapse or one of many threatening complications, as intestinal hæmorrhage,

perforation, venous thrombosis, emboli of the lung, etc., although they may be without any significance, probably through irritation of the intestinal ulcer.

For the especial diagnosis of the febrile diseases, it is necessary to recognize the *type and the course* of the fever; for this purpose, during the entire time of the presence of fever, the temperature is taken at regular intervals and arranged on a fever chart (see below); by this means the *fever curve* is obtained. Many of the acute febrile diseases have characteristic curves.

The *type* of the fever is ascertained by the differences between the morning and evening temperatures.¹ These may be: *continuous* fever in which the day's difference is not more than 1°; *remittent* fever, with a daily difference of more than 1°; *intermittent* fever, in which the fever lasts only a few hours, with an absence of fever during the rest of the day (attack of fever and interval of freedom from fever).

In the course of almost all the febrile diseases, three stages may be distinguished: the time during which the temperature continues to rise (*stadium incrementi*); the *fastigium*, or acme, the period of little change in the temperature, which is usually high; *defervescence* (*stadium decrementi*), the period of decreasing temperature. The decrease may come rapidly in a few hours: *crisis*. The crisis is often heralded by a diminution of the frequency of the pulse and an outbreak of perspiration²; or it may be preceded by a short, very high rise of temperature occasionally accompanied by delirium (*perturbatio critica*); not rarely the crisis is followed by (*epicritical*) delirium; sometimes collapse ensues. The slow decrease of fever extending over days is known as *lysis*.

¹ In practice, the difference between the highest and lowest temperatures taken on the same day; the temperature is usually taken, for one reason or another, more than twice daily.

² The sweat of the crisis is frequently of peculiar, not disagreeable, odor.

The course of the acute infectious diseases, especially those characterized by exanthemata, is divided as follows: (1) stage of *incubation*: from the time of contagion to the beginning of the phenomena of the disease; (2) *prodromal* stage: from the beginning of the fever to the breaking out of the eruption; (3) *eruptive* stage; (4) stage of *desquamation* or defervescence.

The character of the fever. In severe fever, *febris stupida* and *febris versatilis* are distinguished. The former is marked by apathy, a dull, listless countenance, absolute quiet; the latter by an anxious expression, jactation, a mild delirium, picking at the bed-clothes. The transition from the former to the latter state is of evil significance.

Pathognomonic symptoms. After the reading of the temperature, or after the recognition of the fever curve, other quickly appreciable signs which aid in establishing a diagnosis must be looked for. It is well to accustom oneself to a certain routine. (It is best first to inspect the face and skin, then the other organs from above downward.)

1. *Exanthemata.* Characteristic eruptions appear in measles, scarlatina, typhoid fever, typhus fever, variola, varicella, erysipelas. The eruption decides the diagnosis. The exanthemata do not always appear at the beginning of the fever and often disappear before the fever, so that their aid must sometimes be foregone.

2. *Involvement of the sensorium.* Deep apathy is characteristic of typhoid, meningitis, miliary tuberculosis, severe forms of sepsis, and ulcerative endocarditis. Delirium is of no value for the purposes of differential diagnosis.

3. *Herpes labialis and nasalis* (small blebs with serous contents, at angle of the mouth and on the nose, which soon dry up and leave a brownish crust). Herpes is very common in epidemic meningitis and pneumonia, and speaks against tuberculosis, typhoid, and pleurisy.

4. *Frequency of the pulse* under certain circumstances may be of great diagnostic importance. Very slow at the

beginning of meningitis. In scarlatina unusually rapid. In typhoid, aids in the diagnosis of the stage of the disease; in the first stage, comparatively slow, usually not over 110 in uncomplicated typhoid; in the third stage usually from 100 to 120.

5. *Involvement of other organs.* *Lips:* fuliginous (sooty color) in typhoid. *Tongue:* raspberry-like in scarlatina, coated in typhoid. *Neck:* characteristic affections in angina and diphtheria. *Stiffness of the neck* in meningitis. *Rusty sputum* in pneumonia. *Distended abdomen* painless to the touch, in typhoid; retracted abdomen in meningitis. *Enlargement of the spleen* particularly important in typhoid and malaria (Chap. IV.). *Diarrhœa* of characteristic type in typhoid, dysentery, cholera. Redness and swelling of several *joints* in acute articular rheumatism. Condition of the *urine:* diazoreaction of the *urine* in typhoid, etc. Condition of the *blood:* in the majority of the acute infectious diseases the number of the white blood cells is increased (*infectious hyperleucocytosis*); this is absent in typhoid, malaria, glanders, and in many cases of septicæmia.

In many cases it will be possible to make an early diagnosis of the infectious disease present from the recognition of the type of the fever and the consideration of the general and special symptoms.

Yet it must not be forgotten that several days' observation is required for an insight into the course of the fever and that many characteristic symptoms do not develop until the disease is well under way (e.g., exanthemata, enlargement of the spleen, the diazoreaction, diarrhœa, etc.). It is often necessary, therefore, judging from the temperature and the habitus of the patient, to content oneself with the temporary diagnosis "acute infectious disease" and to institute the necessary general therapeutic measures (rest in bed, comfortable position,

light bed-clothing, cool fluid diet, ice-bag to the head, diluted acids, trained nursing). These therapeutic directions are independent of any special diagnosis for a few days, when it is usually possible to make a differential diagnosis from the developed symptoms.

SYMPTOMS OF THE ACUTE INFECTIOUS DISEASES

I. Acute Infectious Diseases with Regular Course of Fever

Measles (*morbilli*) (Fig. 1).—Incubation 10 days, with coryza, cough, gastric disturbances. Prodromata, 2 to 3 days, beginning with chills and high fever. On the 2d or 3d day, diminished fever; on the 3d or 4th day, appearance of the measles eruption, with high temperature. From the 4th to 7th days, continuous fever; on the 7th day, crisis, sometimes lysis. Desquamation for about 14 days, in small scales (furfuraceous desquamation).

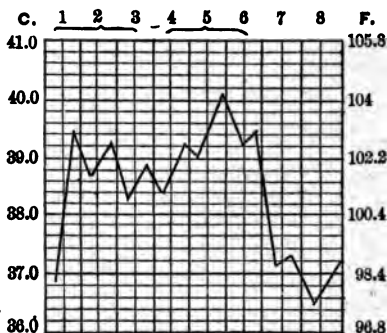


FIG. 1. — FEVER CURVE IN MEASLES.

Further principal symptoms: coryza, cough, conjunctivitis, and photophobia. Pulse somewhat accelerated (in children from 140 to 160). Rare but dangerous complication: broncho-pneumonia. The rare complications with diphtheria and croup suggest a grave prognosis.

Scarlet fever (scarlatina) (Fig. 2).—Incubation, 2 to 24 days, usually without symptoms; prodromata, 1 to 2 days, beginning with chill and high fever. On the 2d day, the eruption of scarlet-red color, with rising temperature. From

the 4th day, lysis. Desquamation, 4 to 14 days; large scales sometimes fall off (*desquamatio membranacea*). Sometimes

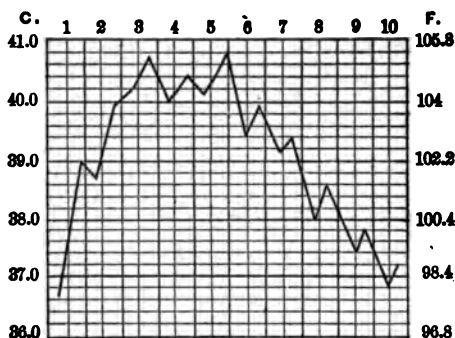


FIG. 2. — FEVER CURVE IN SCARLATINA.

the fever of scarlatina is in no way typical.

Further important symptoms: angina, raspberry tongue, vomiting during the prodromal stage. *Complications and sequelae:* acutenephritis, otitis, more rarely glandular suppuration, joint affections (usually benign), endocarditis.

Erysipelas (Fig. 3). — Incubation, 1 to 8 days. Begins with chill and high fever. On the 1st and 2d days, redness and swelling of the skin. Continuous fever during the extension of the erysipelas. The redness and swelling frequently appear in batches, calling forth irregular remittent or intermittent fever.

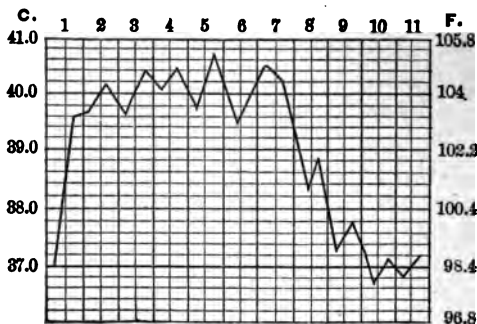


FIG. 3. — FEVER CURVE IN ERYSIPELAS.

The redness and swelling are often confined to the face or to the hairy part of the head (*erysipelas capitis et faciei*), but may extend to the neck and trunk. Erysipelas may follow an injury in any part of the body.

Croupous pneumonia (Figs. 4 and 5).— Begins with chill, high fever, and pain in one side of the chest. Continuous fever. Crisis between the 3d and the 11th days, often on an odd day of the disease, most frequently on the 5th and 7th. Crisis on the 3d day deceptive (Fig. 5); usually followed by renewed continuous fever. Sometimes the crisis is extended over several days (protracted crisis).

Pathognomonic sign: rusty sputum, although some patients with pneumonia have no expectoration, or a whitish-yellow sputum which is not characteristic.

Enlargement of the spleen, which does not disappear until complete resolution has taken place.

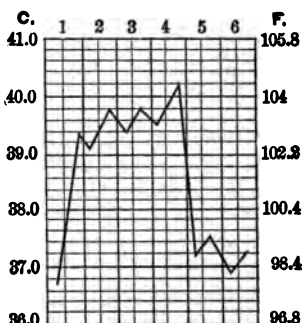


FIG. 4.— FEVER CURVE IN PNEUMONIA. CRISIS 4TH TO 5TH DAY.

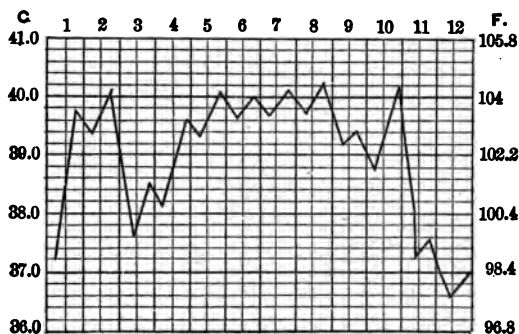


FIG. 5.— FEVER CURVE IN PNEUMONIA. PSEUDOCRISIS ON THE 3D, CRISIS ON THE 11TH DAY.

A new rise of temperature, indicating an advancing infiltration, may follow the crisis. This is called *pseudocrisis*. This is to be diagnosticated, when in spite of the fall of temperature, the pulse

and rapidity of the respiration remain abnormally high; or when, despite the normal temperature, the hyperleucocytosis persists.

Physical signs of the infiltrated lung at the height of the disease: dulness (with a tympanitic note) and bronchial breathing (cf. Chap. VI.).

If a regular crisis does not supervene, or if the fever rises irregularly after the crisis, it is to be referred to a pleurisy (serous or purulent), more rarely gangrene, tuberculosis, or abscess of the lung.

Typhoid fever (Fig. 6).—Incubation 7 to 21 days. Prodromata for about a week, with indefinite symptoms of

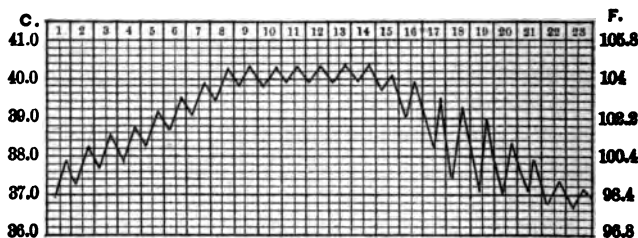


FIG. 6.—SCHEMATIC FEVER CURVE IN TYPHOID FEVER.

general lassitude. *Stadium incrementi*, terrace-like rise of temperature of remittent character as it ascends. Acme reached on 4th to 7th day. *Fastigium*, continuous fever. *Defervescence*, fever remitting as it descends. Morning temperatures sink daily; during the first days the evening temperatures reach a high point (uncertain stage, steep fever curve).—The duration of the 2d stage varies with the severity of the disease. In light cases the descending remission of the fever may begin on the 16th or 14th days or even earlier; in very severe cases the continuous fever may last until the 5th week.

Further principal symptoms: apathy, stupor, fuliginous, furred tongue. Roseola from the end of the 1st to the middle of the 2d week. Enlargement of the spleen during the

acme. Meteorism. Stools, diarrhoeal of pea-soup consistency. Diazoreaction in the urine, no hyperleucocytosis. Frequently, bronchitis.

The diagnosis is made by the presence of several of these signs at the same time; it is scarcely possible to do so from any single one. Many symptoms may be absent; for instance, decided meteorism, the diazoreaction, the diarrhoea.

The diagnosis of typhoid must embrace the week and stage of the disease, if possible. This is sometimes facilitated by complications which mark certain periods; thus, *intestinal hæmorrhages* take place most frequently during the time of defervescence; and during this period, too, fatal perforation of the intestine sometimes occurs (vid. p. 100).

The prognosis depends, among other things, upon the frequency and tension of the pulse and the degree of stupor.

Typhus fever (*typhus exanthematicus*, spotted fever) (Fig. 7).—Incubation 3 to 21 days. No prodromata. Begins

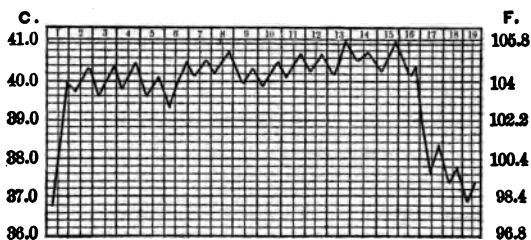


FIG. 7.—FEVER CURVE IN TYPHUS FEVER.

with chill and high temperature. On the 3d day, a profuse roseola. Continuous fever from 13 to 17 days with slight remissions on the 6th to the 8th day. Temperature falls by crisis, with delirium.

The roseola soon becomes petechial. Bronchitis. The mental symptoms especially severe.

Recurrent fever (relapsing fever) (Fig. 8).—Incubation 5 to 7 days. No distinct prodromal stage. Begins with

chill and rapid rise of temperature. Continuous fever 5 to 7 days. Temperature falls by crisis. Then 5 to 8 days' absence

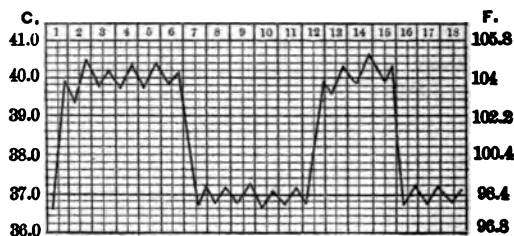


FIG. 8.—FEVER CURVE IN RELAPSING FEVER.

of fever; thereupon renewed continuous fever, usually of short duration. Frequently after absence of fever for 7 days, continuous fever for from 2 to 3 days. — Great rapidity of pulse. Enlargement of the spleen. Roseola. Herpes. *Spirochetæ Obermeieri* in the blood during the fever (Chap. XII.).

Variola (small-pox) (Fig. 9).—Incubation 10 to 13 days. The actual course of the disease may be divided into 4

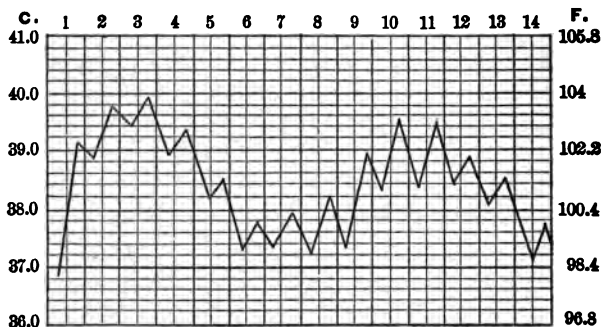


FIG. 9.—FEVER CURVE IN VARIOLA.

stages: *stage of invasion* (prodromata), beginning with chill and high fever, 3 to 4 days. *Stage of eruption*, diminished

fever to the 9th day. *Stage of suppuration* (fever of pus), severe remittent fever, 9 to 11 days. *Stage of exsiccation* (drying up of the papules), fall of the temperature by lysis.

The eruption forms at first red spots which gradually change into larger papules. On the 6th day they become filled with a cloudy fluid, on the 8th day they are blebs filled with pus; from the 9th day the contents are poured out, and the drying up begins on the 11th day. The eruption attacks the mucous membrane of the mouth and throat. — Severe drawing pains in the knees and back.

Varioloid (Fig. 10) is the mildest form of small-pox as it is seen, after contagion, in persons who were unsuccessfully vaccinated or vaccinated more than ten years previously. In varioloid, the period of invasion is followed immediately by the stage of drying up, without the fever of suppuration. The eruption may be merely indicated or it may appear in some irregular form (erythema).

Varicella (chicken-pox). — Fever begins with a chill and is continuous until the drying up of the eruption, 2 to 4 days.

Eruption characteristic: rose-colored, slightly-elevated spots, which soon become vesicular. Found on the gums and throat also. Seldom like variola, but then to be distinguished by the fact that in varicella all the stages of the eruption appear simultaneously. Prognosis absolutely good. In very rare cases sequelæ are seen; for instance, acute nephritis.

Malaria (intermittent fever) (Figs. 11, 12, 13). — Incubation, 7 to 21 days. No distinct prodromata. Chill with high

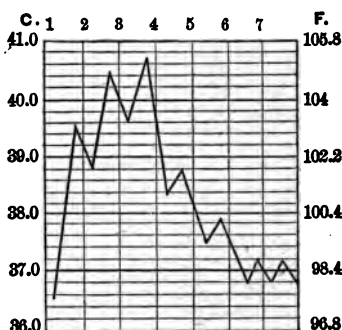


FIG. 10. — FEVER CURVE IN VARILOID.

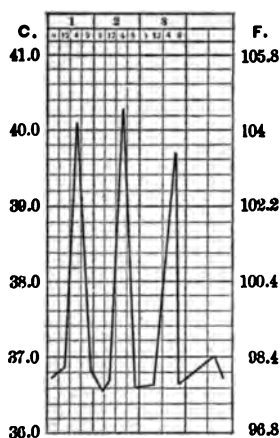


FIG. 11. — FEVER CURVE IN QUOTIDIAN INTERMITTENT FEVER.

elevation of temperature; in a few hours, critical descent of fever, with perspiration, then apyrexia. Shortly before and during the fever, the cause of the malaria, the *plasmodium of Laveran*, in the blood. The attack is repeated at the same time of day the following day (quotidian type), Fig. 11; or on alternate days (tertian type), Fig. 12; or every fourth day (quartan type), Fig. 13. If the introductory chill appears before or after the regular time of day, it is spoken of as anticipated or postponed intermittent fever.

Two attacks in one day characterize double intermittent fever.

The diagnosis of malaria is rendered absolutely certain in doubtful cases by the microscopic demonstration of the plasmodium in the blood; also by the establishment of the type of the fever, the enlargement of the spleen, and the specific controlling action of from 1 to 2 grammes of quinine when this dose is administered 6 hours before the expected attack of fever.

In the tropics there are forms of malaria in which the fever is entirely irregular; at the same time organic affections appear,

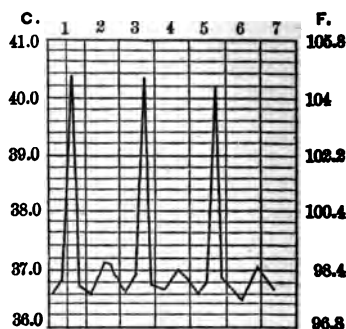


FIG. 12. — FEVER CURVE IN TERTIAN INTERMITTENT FEVER.

which totally obscure the picture of the disease (masked intermittent fever). The diagnosis is made certain only by the curative action of quinine.

In the temperate climates, *malarial neuralgia* is seen, attacks of neural pain which return at certain times of the day like the paroxysms of fever. They are controlled by quinine (*e.g.*, supraorbital neuralgia).

Irregular fever, intermittent in type, which does not yield to quinine, and in which the malarial plasmodia can not be found in the blood, is to be referred to *deeply lying abscesses* or *endocarditis* or *latent tuberculosis*.

Influenza: after a short prodromal stage, sudden, usually high fever, which lasts for several days in continuous and remittent form, accompanied by intense prostration and great pain in the extremities. Frequently no localization, frequently catarrh of the bronchi. The unusually large number of complications or sequelæ is characteristic: respiratory tract (catarrhal and croupous pneumonia); circulatory apparatus (endocarditis, thrombosis); nervous system (neuralgias and psychoses), etc. The prognosis is good except when complications or sequelæ set in.

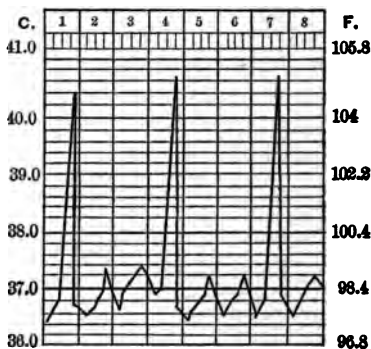


FIG. 18. — FEVER CURVE IN QUARTAN INTERMITTENT FEVER.

II. Acute Infectious Diseases without Regular Course

A number of acute infectious diseases run their courses with irregular fever which does not conform to any type. The diagnosis of these diseases rests upon the local lesions.

Follicular amygdallitis (*angina follicularis*). — Redness and swelling of the soft palate, the tonsils, and the pharynx, often accompanied by a white or grayish exudate, which can

usually be removed without causing hæmorrhage. Strep-tococci are most often found in the membrane. Submax-illary glands frequently involved. Fever, beginning with a chill, continuous for several days or lightly remittent. General disturbances not very severe despite the high temperature; if severe, diminished in a few days. An acute nephritis may appear after a light attack of angina.

Abscess of the tonsil sometimes supervenes (*angina apostematosa*).

Diphtheria. — Tonsils and palate covered with a grayish-white membrane, the removal of which produces bleeding from the mucous membrane. Diphtheria bacilli may be obtained from this membrane by culture. In severe cases, the membrane involves the nose, the larynx, the bronchi. Enlargement of the submaxillary glands. The profound general disturbance is characteristic (small, rapid pulse, stupor). Frequently, albuminuria. Fever throughout atypical, and gives no data for the prognosis, which depends in part upon the severity of the local affection, in part upon the profundity of the general infection. Frequent characteristic sequelæ: paralysis of accommodation, of the palate, of the extremities. There are cases of pharyngeal inflammation, in which the diagnosis between diphtheria and angina can not be made for some time; the decision must be reached by a bacteriological examination (cf. Chap. XII.).

Acute miliary tuberculosis. — Atypical fever. Marked cyanosis and dyspnœa. Over extensive areas of the lungs crepitant râles without dullness. In the urine, diazoreaction. In some cases tubercles in the choroid may be found ophthalmoscopically. Prognosis very bad; death in from 8 to 14 days.

Cerebro-spinal meningitis. — Irregular, partly remittent, partly continuous fever, with a long course and many remis-

sions. Deep stupor. Pathognomonic symptom: stiffness of the neck. In the early stages, hyperæsthesia of the extremities, slow pulse.

The diagnosis must include the etiology. 1. *Epidemic* (sporadic) meningitis is diagnosticated by positive exclusion of other causes; during an epidemic the diagnosis is easier. 2. *Tubercular* meningitis may be diagnosticated in the presence of tuberculosis of the *lungs*, usually somewhat advanced. Herpes labialis never present. Recently this diagnosis has been repeatedly made by finding tubercle bacilli in the cerebro-spinal fluid obtained by puncture of the cerebro-spinal canal (lumbar puncture of *Quinke*). 3. Meningitis due to an extension of an *otitis media*.

The *stiffness of the neck* is usually not very distinct during the first few days, but is well elicited by the end of the first week. If the meningitis advances, the stiffness disappears, but the symptoms of paralysis supervene. A mild stiffness of the neck may be present at the beginning of severe cases of pneumonia and typhoid; meningitis may accompany these diseases.

Acute articular rheumatism.—Irregular remittent fever. Redness, swelling, and pain in several joints, usually affecting both sides of the body symmetrically. Fever and local affection yield in two-thirds of all cases to salicylic acid or antipyrin. Frequent complications: endocarditis, with permanent valvular lesions; more rarely, pleurisy, pericarditis. Atypical forms of articular rheumatism, especially in gonorrhœa.

Parotitis epidemica (mumps).—Irregular, moderately high fever, lasting from 2 to 8 days. Pain, enlargement of one or both parotids; resolution usual, sometimes suppuration. In a few cases, accompanied or followed by a febrile orchitis, more rarely epididymitis.

Sepsis (pyæmia, septicæmia, blood poisoning).—Atypical, usually remittent fever, commonly with irregular chills. Stupor, great prostration. Usually no hyperleucocytosis. Primary suppuration often present, as, infected wounds, in the uterus (puerperal sepsis), in paronychia and phlegmo-

nous processes, in cavities of the head (empyema of the antrum of Highmore, otitis media), in the prostate. The entrance of the producers of the process may remain hidden (cryptogenetic septicæmia). Shortly before death the responsible bacteria may be found in the blood. *Pyæmia* is characterized by localized abscesses in various organs of the body, while *septicæmia* denotes the general toxæmia without localization. These two forms of disease frequently pass over into each other. In the pure pyæmic type, hyperleucocytosis usually exists. Differentiation between the various kinds of sepsis according to the etiology (streptococci, staphylococci, diplococci, etc.) has no clinical value.

Acute endocarditis. — Irregular, usually remittent fever, with the physical signs of a lesion of a cardiac valve (intracardiac murmur; the systolic murmur alone does not denote an endocarditis). The diagnosis must differentiate between endocarditis of benign (verrucous or warty) and of malignant (ulcerative) character. The latter diagnosis is certain when repeated chills and many cutaneous emboli manifest themselves. A benign endocarditis frequently becomes malignant. An acute endocarditis develops during or after other infectious diseases. Endocarditis following articular rheumatism is usually warty, and disappears, leaving valvular lesions. Endocarditis accompanying sepsis, pneumonia, gonorrhœa, usually ulcerative.

CHAPTER III

DIAGNOSIS OF THE DISEASES OF THE NERVOUS SYSTEM

THE following data are especially important in the anamnesis and history of nervous diseases: (1) *Heredity* in psychical diseases, neurasthenia, epilepsy, hysteria; syphilis in the parents. (2) Previous disease, especially syphilis, acute infectious disease. (3) Causes and predisposing factors: trauma, exposure, fright, intoxications (lead, mercury, alcohol, excessive use of tobacco).

The serial data for registering the *status præsens* are to be found on page 2, under B. I. The disturbances of sensation and motility require special consideration (B. I. 6), for which the following guide or scheme is in use at the 1st Medical Clinic of Berlin:—

STATUS OF NERVOUS SYSTEM

A. MOTION

I. *Position of the limbs at rest.*

- a. Abnormalities in position.
- b. Atrophy?
- c. Abnormal involuntary movements (tremor, convulsive movements)?

II. *Movements.*

- a. Free, active.
- b. With resistance, active.
- c. Passive.

1. Face

I. Are both halves symmetrical?

Equal palpebral fissures?

Equal nostrils?

Mouth, straight or crooked?

Are the eyes normal or quiet?

Pupils equal?

- II. Request the patient to wrinkle the forehead, close the eyes, pucker the mouth, laugh, blow out the cheeks, extrude the tongue, move it to right and left, move the eyes (to the right, left, upwards, downwards, and to converge).

2. Cavity of the Mouth ; the Larynx

- I. Condition of the soft palate and uvula.
II. Elevation of the same in phonation.

3. Throat and Neck

- I. Position of the head.
II. Turn head to right and left, bend it forwards and backwards (oppose resistance lastly).

4. Shoulders and Arms

- I. Appearance of the scapula, position of the arms, of the fingers, size of the thenar and hypothenar eminences, condition of interosseous spaces.
II. Elevate the shoulders, raise the arms (to the vertical position), abduct them, hold them horizontally forwards.
Flex and extend the forearm.
Pronate and supinate.
Flex and extend the fingers, spread them apart, close the thumbs upon the palms.
Force of grasp.

5. Legs

- I. Condition of the trochanters.
II. Elevation, abduction and adduction.
Flex and extend the legs.
Flex and extend the feet.

6. Trunk

- I. Breathing (rhythm)?
Abdomen (retracted)?
Spinal column (shape)?
Buttocks (hypertrophied or atrophied)?
II. (Only necessary if a disturbance is perceived during inspection).
Bend trunk forwards, backwards, to the sides, take deep inspiration, cough.

7. Bladder and Rectum (inquiry)
8. Equilibrium with closed eyes
9. Gait
10. Ability to grasp objects
11. Speech
12. Chirography

B. SENSATION

- I. *Subjective signs*: deafness, formication, pain, etc.
- II. *Objective examination*.

- a. Sensibility of the skin.
 1. Most gentle contact.
 2. Painful pin pricks.
 3. Cold.
 4. Heat.
- b. Muscular sense.
 1. Sensation of movement.
- c. Visual field.
- d. Hearing.
- e. Smell.
- f. Taste.

C. REFLEXES

- a. Skin reflex.

Reflex of sole and palm, cremaster, abdominal, lids, conjunctiva, palate.
- b. Tendon and periosteal reflex.

Patella, tendo Achillis, foot clonus, wrist, biceps, triceps.
- c. Pupillary reflex.

Condition of the sensorium: dulness, stupor, apathy in febrile diseases is an indication of an intense infection, and suggests a grave prognosis. These mental states are of diagnostic value in typhoid, meningitis, miliary tuberculosis, sepsis.

In non-febrile diseases apathy and stupor are, as a rule, the precursors of complete unconsciousness (*coma, lethargy*).

Coma is present in cases of: 1. Poisoning; in these cases the history will determine the condition, or, better, the examination for poison of the contents of the stomach obtained

by lavage. 2. Apoplexy; often recognized by the coincident facial palsy, or by a hemiplegia; the deeper and the longer in duration the coma, the worse the prognosis. 3. In increased cerebral pressure, for example, induced by tumors, associated with choked disc. 4. In the course of the diseases due to perverted or defective metabolism, especially diabetes; recognized by the deep and laborious respiration (intense breathing of *Kussmaul*). Usually lethal in termination. It is seldom present in carcinoma and anæmia. 5. During the course of nephritis (*uræmic coma*), commonly preceded by repeated attacks of convulsions and high arterial tension. An important diagnostic rule may be thus derived: in every case of coma arising from an unknown cause, the urine should be carefully drawn by catheter and examined for both *albumin* and *sugar*. Markedly rigid radial arteries would point to cerebral apoplexy. In many cases the diagnosis will be determined by the history obtained from the relatives and friends.

Dulness of the sensorium, ranging from slight apathy to stupor, is often a sign of an existing *psychosis*, which could be diagnosed by the presence of hallucinations and delusions, but better by the exclusion of all the above-mentioned visceral causes of coma.

It is especially important for the diagnostician to recognize the *psychoses* due to certain drugs (salicylic acid, bromides, etc.), to chorea, to certain of the last stages of heart affections, to all conditions of inanition; in all these there are illusions and hallucinations of hearing and sight, as well as delusions of persecution and those of a religious character.

Essential mental disturbances (mania, melancholia, paranoia, parietic dementia) in well-developed cases are readily recognized, and would most likely be turned over to asylums (strict legal forms being observed). Most frequently the first indistinct beginning of dulness of judgment or of perception succumbs to the acumen of the physician.

To determine such indefinite psychical anomalies (motiveless pleasurable and painful feelings, sudden change of mood, diminution of intelligence and of memory, moral decrepitude) the physician must carefully investigate whether the case is one of psychic

reaction to a somatic functional disturbance or of a beginning insanity. In the latter case the signs of an impending paralytic dementia ought to be looked for: iridoplegia, inequality of the pupils, absence of or increase of the knee jerk, disturbances of speech, etc. The further elucidation of this most highly important question would be transferred to the psychiatrist.

Delirium in febrile diseases is conditional to the height of the fever and of the intoxication; it is without essential prognostic significance. It occurs frequently before the crisis of the disease (*perturbatio critica*), after its crisis (delirium of defervescence), and it is often the sign of collapse. When accompanying non-febrile affections (irrespective of the psychoses and intoxications, especially alcoholism), it is always an indication of intense exhaustion (inanition, delirium).

Diffuse headache when *transitory* is usually of small diagnostic importance (as in fever, over-exertion, excesses, dyspepsia, constipation, female genital affections, etc.). *Paroxysmal* headache is often confined to *one side* of the head. *Migraine*: the latter is chiefly associated with dyspeptic signs, and often terminates in an attack of vomiting; not infrequently is it limited to the course of distribution of certain nerve trunks; when occurring thus, painful points on pressure may be elicited. *Neuralgia*: continuous headaches occur in neurasthenia as well as in chlorosis, heart diseases, etc.; when occurring with diabetes it denotes a grave form of the disease. *It is often the first sign of a developing uræmia*. It also occurs in tertiary syphilis, and is then especially nocturnal (*dolores osteocopi*).

Vertigo. Occurs frequently in gastric and intestinal diseases. When present in a heart affection it points chiefly to a stenotic lesion. It accompanies anæmia. When concomitant with shrill tinnitus aurium, it indicates an aural disease (labyrinth), and is known as the *Ménière* symptom. It is also present in cases of cerebral tumors and special

affections of the cerebellum. It is not infrequently associated with imperative movements.

ANATOMICAL INTRODUCTION

An intimate knowledge of the anatomy of the central nervous system is essential in order to form a correct diagnosis of the various forms of disease of the nervous system.



FIG. 14.—EXTERNAL SURFACE OF THE LEFT CEREBRAL HEMISPHERE.
(Drawn from a brain treated with nitric acid and dried)

- | | | | |
|---|------------------------------|----------------|------------------------|
| a | central fissure | B | post-central gyrus |
| b | pre-central fissure | C ₁ | superior frontal gyrus |
| c | parieto-occipital fissure | C ₂ | middle frontal gyrus |
| d | intraparietal fissure | C ₃ | inferior frontal gyrus |
| e | Sylvian fissure | D | parietal lobe |
| f | superficial temporal fissure | E | marginal gyrus |
| g | second temporal fissure | F | angular gyrus |
| h | inferior occipital fissure | G | occipital lobe |
| i | anterior occipital fissure | H ₁ | first temporal gyrus |
| A | pre-central gyrus | H ₂ | second temporal gyrus |

The most important anatomical features which are necessary for clinical work are here briefly presented.

The motor tracts proceed from the psychomotor centres of the cerebral cortex (Fig. 14).

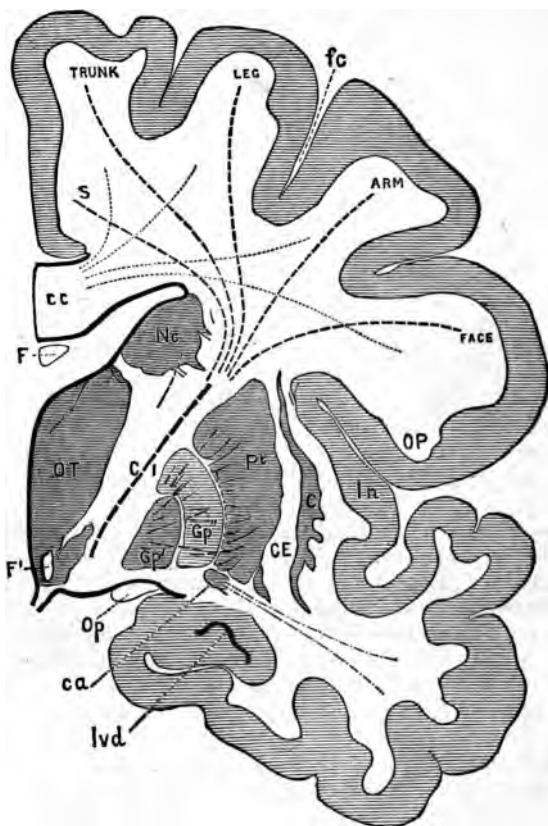


FIG. 15. — OUTLINE OF A TRANSVERSE DORSO-VENTRAL SECTION OF THE RIGHT HALF OF THE BRAIN. (Natural size.) (Sherrington.)

OT, optic thalamus; *Nc*, nucleus caudatus; the head only appears in this section; *Pt*, putamen; *Gp*, *Gp'*, the two parts of the globus pallidus of the nucleus lenticularis; *C*, the claustrum; *CE*, the external capsule; *In*, the island of Reil; *ca*, the anterior commissure shaded to render it distinct, and the fibres from the temporo-sphenoidal lobe which pass into it being indicated by broken lines; *Op*, the optic tract; *lvd*, the end of the descending horn of the lateral ventricle; *F*, the fornix; *F'*, the end of the anterior pillar of the fornix in the base of the thalamus; *cc*, corpus callosum; *OP*, anterior part of the occipital lobe.

fc is the central fissure or fissure of Rolando. The course of the fibres of the pyramidal tract connected respectively with the trunk, leg, and arm, and hence with spinal nerves, and of those connected with the face and hence with cranial nerves, is shown by broken lines. These are all seen converging into the internal capsule, *CI*.

The centre for the voluntary movements of the arm lies in the middle third of the pre-central convolution; the centre for the face and tongue in the lower third of the same convolution; that for the motion of the legs, in the upper third of both the pre-central and post-central gyri and in the para-central lobule, which unites these two convolutions on the mesal face of the hemisphere. The motor centres of speech lie in the posterior part of the left inferior (third) frontal gyrus and in the insula (island of Reil) (between the inferior frontal and the superior temporal gyri). The sensory speech centre lies in the temporal lobe (first left temporal gyrus). The cortical visual centre is situated in the occipital lobe (for hemianopsia see p. 38, cerebral nerves), the cortical auditory centre in the temporal lobe.

The motor fibres proceed from the cortical centres through the *corona radiata* to the *internal capsule* (Fig. 15).

Here will be found the pyramidal (motor) tract in the middle third of the posterior half of the internal capsule, and between the thalamus and lenticular nucleus, closely bordering on the fibres which control voluntary facial movements. This location is the seat of predilection for cerebral hæmorrhages.

Proceeding through the internal capsule, the motor fibres continue to the *pes* (ventral portion of *crus cerebri*); the sensory fibres proceed through the tegmentum (dorsal portion of *crus cerebri*) into the *pons*. From the *pons* they go into the *medulla (oblongata)*, where they constitute the *pyramids*, and there decussate for the most part. In the *spinal cord* (Fig. 16) the decussated motor fibres proceed downwards in the *crossed pyramidal tract*, the very few fibres which did not decussate proceeding downwards as the *uncrossed (anterior) pyramidal tract*.

The motor fibres proceed from the pyramidal tracts to the *anterior cornua* of the central gray matter of the spinal cord, and from there leave the cord by way of the *anterior spinal roots* and the peripheral nerves to reach the muscles.

The trophic centres for the pyramidal tract lie in the cerebrum, and when any part of the motor tract is injured there ensues, in addition to the consecutive paralysis, a descending degeneration of the pyramidal tract; the trophic centres for the peripheral *motor nerves* lie in the ganglion cells of the anterior cornua. A lesion of this organ or one peripheral to it induces degeneration of the nerves and paralysis and atrophy of the muscles supplied by the structures concerned.

The *sensory tracts* run upwards in the *posterior columns* and

posterior cornua of the cord; they decussate immediately after their entry and also in the lemniscus.

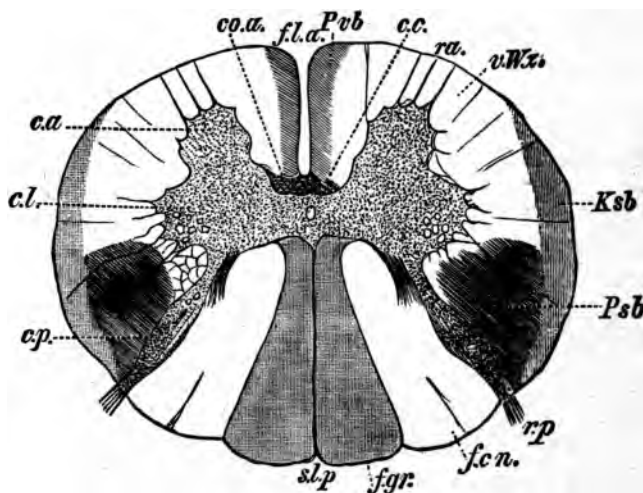


FIG. 16.—DIAGRAMMATIC CROSS-SECTION OF THE SPINAL CORD SHOWING THE TRACTS OF THE WHITE MATTER.

<i>c.a.</i> (<i>cornu anterius</i>) anterior horn	<i>f.gr.</i> (<i>funiculus gracilis</i>) Goll's column
<i>c.l.</i> (<i>cornu laterale</i>) lateral horn	<i>f.cn.</i> (<i>funiculus cuneatus</i>) Burdach's column
<i>c.p.</i> (<i>cornu posterius</i>) posterior horn	<i>Ksb</i> (<i>Kleinhirnsseitenbahn</i>) direct cerebellar tract
<i>c.c.</i> central canal in the gray commissure	<i>Psb</i> (<i>Pyramidalseitenbahn</i>) crossed pyramidal tract
<i>co.a.</i> anterior white commissure	<i>Pvb</i> (<i>Pyramidalvorderbahn</i>) direct pyramidal tract
<i>ra.</i> (<i>radix anterior</i>) anterior root	<i>v.W.x.</i> (<i>Vordere Wurzelfasern</i>) anterior radicular zone
<i>rp.</i> (<i>radix posterior</i>) posterior root	
<i>f.l.a.</i> (<i>fissura longitudinalis anterior</i>) anterior fissure	
<i>sl.p.</i> (<i>septum longitudinale posterius</i>) posterior septum	

PARALYSIS

(The absolute inability to move a limb is called *paralysis*; to motor weakness of a limb is given the name *paresis*.)

When the presence of a paralysis or paresis is determined, the following features should be elicited:—

1. Is the paralysis limited to one side of the body (*hemiplegia*, for example, paralysis of the right arm and

right leg); does it involve both sides (*paraplegia*, e.g., both arms or both legs); or is it limited to one extremity or to a group of muscles (*monoplegia*, e.g., one arm, or the servati group)? All hemiplegias are produced by lesions of the cerebrum, paraplegias by lesions of the spinal cord, although multiple neuritis occasionally is the cause of the latter. Monoplegias depend upon lesions either of the cerebrum or of the peripheral nerves.

2. Is the affected member *flaccid* or *spastic*?

Flaccid paralyzed limbs are easy to move by passive motion, but a spastic condition of the limb presupposes an extensive resistance to any attempts of motion, or a convulsive contraction ensues on such attempt. The reflexes in the latter condition of spastic paralysis are increased in the affected limb.

Spastic conditions ensue when contracture develops in the paralyzed muscles or in their antagonists, and further in those conditions of increased reflexes like those accompanying degeneration of the pyramidal tract.

3. Does *atrophy* exist? Atrophic palsies depend upon lesions of the peripheral nerves or of the anterior cornua of the spinal cord, or of the cranial nerve nuclei or of the pons.

The organic paralyzes are in direct contrast to the *functional*, which do not depend upon anatomical causes but upon an affection of volition (hysterical paralysis, paralysis of fright). The functional palsies are recognized by the following criteria: there is an absence of trophic and electrical disturbances; there are present and coincident with the palsy certain definite signs of hysteria, such as hemianæsthesia, convulsions, contractures, etc.; above all they may be recognized by the general psychical character of the patient.

HEMIPLEGIA

It is important in this condition to determine the etiology and to locate the part of the cerebrum which is involved in the process producing this symptom (localization).

Etiology

1. *Embolism.* The hemiplegia is suddenly produced, involves chiefly the Sylvian artery, and is thereby associated with motor aphasia. It is necessary to determine the origin of the source of the embolism, *e.g.*, affections of the left side of the heart.

2. *Apoplexy.* It appears suddenly, and is proven by an existing arterio-sclerosis or of contracted kidney. It involves chiefly the large central ganglia (corpus striatum, internal capsule) (Fig. 15), and occurs frequently without aphasia. It is usually accompanied by a long-continued comatose condition.

3. *Syphilis.* It may be the result of a focal softening induced by the closure of an artery whose walls have been the seat of a syphilitic endarteritis. It develops slowly, and is often preceded by prodromal headaches and vertigo; often there develop other focal lesions, manifesting themselves at times in ocular palsies, paralysis of muscles on the other side of the body, disturbance of speech such as anarthria. The proof of an existing or preëxisting syphilis will assist in determining the etiological factor of the hemiplegia. Very often antisymphilitic treatment produces a favorable result.

4. *Simple focal softening due to arterio-sclerosis.* The diagnostic factors in this case are old age, existing arterio-sclerosis, absence of syphilis, no improvement with anti-symphilitic treatment.

5. *Toxic causes.* It occurs with uræmia, in the last stages of carcinoma and phthisis, and is then evanescent and atypical in character.

It is wise to regard tentatively that hemiplegias occurring in the young are of syphilitic basis, and to always subject all those patients in whom there is a doubt as to the etiological factor to antisymphilitic treatment.

Localization of the lesion giving rise to hemiplegia may often be determined by the simultaneous involvement of the cerebral nerves and speech.

The following focal lesions should be chiefly borne in mind: 1. Hemiplegia with motor aphasia indicates a lesion of the third left frontal convolution. 2. Hemiplegia with lower facial palsy, a lesion in the posterior half of the internal capsule. 3. Hemiplegia with hemianæsthesia, a lesion in the posterior division of the same half of the internal capsule. 4. Hemiplegia with alternating (crossed) ocular palsy, a lesion in crus cerebri. 5. Hemiplegia with alternating (crossed) facial paralysis (*Gubler*), a lesion of the pons. 6. Hemiplegia with anarthria and deglutitory palsy, a lesion of the medulla oblongata.

PARAPLEGIA

It is necessary to decide whether the paraplegia is the result of a *lesion of the spinal cord* or of a *peripheral neuritis*. All *spastic* paraplegias may be assigned to spinal cord lesions. The following features must be determined in a *flaccid*¹ paraplegia: 1. Whether the sphincters (bladder, rectum) are involved, a condition which arises only in spinal cord disease. 2. The *reflexes*; they are absent in neuritis, and are exaggerated in spinal cord disease. 3. The etiology; paralysis due to alcohol is of peripheral origin.

In a few individual cases it may be extraordinarily difficult to differentiate between poliomyelitis (a disease of the gray substance of the cord) and a peripheral neuritis, because in exceptional cases the involvement of the sphincters occurs in neuritis; just as the reflexes may in the same manner be exaggerated in the hyperæsthetic stage of a neuritis, so may the reflexes be absent in a deep myelitis which destroys the reflex arc.

¹ May we use this adjective to denote what is expressed in the German "*schlaffen Paraplegien*"? The author means the paraplegia associated with flaccid, limp, or relaxed muscles, as the antithesis to the *spastic* paraplegia.—THE TRANSLATORS.

Should the diagnosis of a cord lesion be made, it is necessary to establish the following data:—

1. The seat and extent of the disease; paralysis of both legs indicates a lesion in the lumbar and lower dorsal segments of the cord; paralysis of both legs and arms, a lesion of the upper dorsal and cervical segments.

2. The nature of the pathological process, whether a myelitis, a tumor (aneurysm very seldom occurs), carcinoma, tubercular caries, or syphilis. A tumor, or caries, must be palpable (the vertebra must be carefully examined), carcinoma and tuberculosis in other organs must be demonstrated in order to establish a positive result. The clinician's thoughts should turn to aneurysm if there be a concomitant arterio-sclerosis or an idiopathic cardiac dilatation; to syphilis if the cranial nerves be also involved, or if a specific infection is conceded.

In all doubtful cases antisyphilitic treatment should be given.

PARALYSES OF THE CEREBRAL AND SPINAL NERVES

Paralysis of the cranial nerves. This is recognized by the defective function of the muscles supplied by them, and can be determined by the anatomical relations (Fig. 17).

The most important signs are here mentioned:—

Olfactory lesion is to be determined by disturbance of the sense of smell, which should be tested by the use of odorous but not irritating substances (musk, asafoetida). Still one must bear in mind the possibility of disease of the nasal mucous membrane and of nasal occlusion.

Optic nerve lesion will be indicated by diminution of vision, limitation of the visual field, color-blindness. (In every case an ophthalmoscopic examination should be made.)

Hemianopsia or *hemipopia* (the loss of sensation in one half of the retina of each side) depends upon lesion of the occipital lobe or of the optic tract as far as the chiasm. The focal lesion is cephalad to the corpora quadrigemina in homonymous hemianopsia when the pupillary light reaction is normal; on the other hand,

should the pupillary reflex be absent for those visual rays which fall upon the paralyzed halves of the retina, the cause of the hemianopsia must be sought ventral to the corpora quadrigemina or in the optic tract. The latter condition is called *hemianopic iridoplegia* or *hemipopic pupillary reaction*. *Amblyopia* or *amaurosis* of one eye

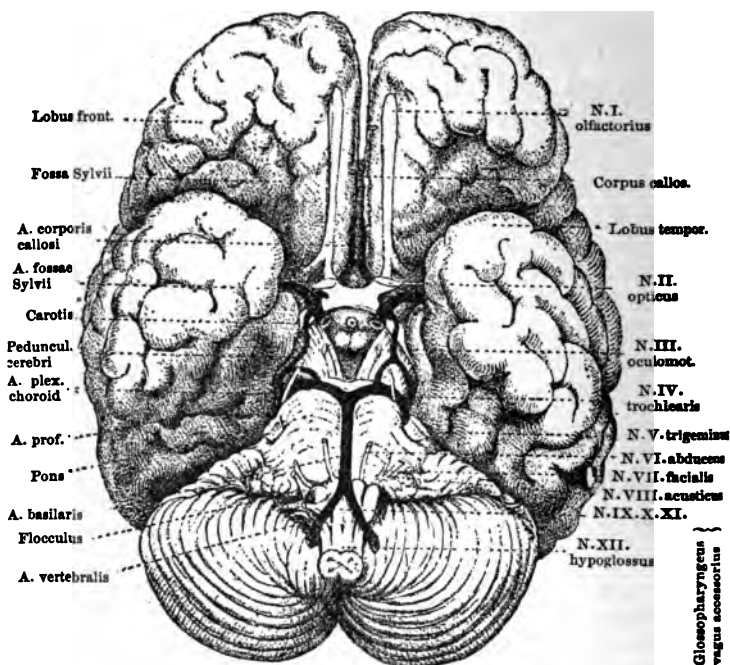


FIG. 17.—THE BASE OF THE BRAIN AND THE CRANIAL NERVES. (AFTER HENKE.)

indicates lesion of the optic nerve anterior to the chiasm; it may also be induced by toxic influences (tobacco amblyopia, uræmic amaurosis).

Motor oculi paralysis is shown by a divergent strabismus, dilatation of the pupil (*mydriasis*), double vision (*diplopia*), closure of the upper eyelid (*ptosis*).

Recurrent ocular motor paralysis accompanies conditions of migraine, which may last for weeks or months (*migraine ophthalmique*).

of Charcot), but in such cases the doubt of an organic cerebral lesion (paretic dementia) should always be entertained.

Mydriasis may also be produced by an irritation of the sympathetic, such as results from migraine, trauma, pressure, diseases of the cervical cord, poisoning from atropine, cocaine, etc. It may be also reflex, as in great fright, severe dyspnoea, intense pain.

Meiosis, contraction of the pupil, depends upon an irritation of the motor oculi or lesion of the cervical sympathetic; it is unilateral in migraine, and may be the result of the pressure of a tumor on the cervical sympathetic; it is bilateral in affections of the cervical cord and in pilocarpine poisoning, etc. Concerning reflex iridoplegia, see p. 56.

Trochlearis paralysis is shown by the inability to move the eyes upwards and outwards.

In *abducens* paralysis the eye can not be moved outwards.

Certain abnormal positions of both ocular globes point towards central focal lesions (associated palsies, *nuclear* palsies); for instance, both eyes are immovably fixed looking to the right.

When the muscles of mastication will not perform their function, it indicates a paralysis of the motor division of the *trigeminus*.

Facial palsy is indicated by a loss of function of the muscles of expression. The exact seat of the lesion which produces the interruption of motor conduction may be determined according as the sense of taste, the salivary secretion, hearing, the uvula may be involved (*Erb's* scheme).

Disturbances of hearing may indicate an *acusticus* affection. An otoscopic examination should, however, decide.

Glossopharyngeal paralysis is indicated by a disturbance of taste in the posterior part of the tongue.

Hypoglossal lesion is shown by the deviation of the tongue to one side or the other.

Spinal accessory lesion produces a paralysis of the sterno-cleido-mastoid and trapezius.

Pneumogastric lesion presents an increase in pulse beat and a slowing of respiration.

Lesions of the nerves of the cord are recognized by the loss of function of the muscles which they supply.

Only the symptom groups which commonly appear are here noticed:—

Erb's paralysis is the paralysis of the deltoid, biceps, brachialis

anticus, supinator longus, and the infraspinatus. It is due to a lesion of the brachial plexus (5th to 8th cervical and 1st to 2d dorsal nerves).

Median nerve lesion. In this affection, it is impossible to pronate the forearm and flex the hand; likewise the flexors of the thumb, and their antagonists, and the flexors of both the last phalanges are involved. The proximal phalanges can be flexed, and the third, fourth, and small fingers are capable of motion.

Ulnar nerve lesion. Flexion and motion of the hand to the ulnar side and flexion of the third finger are disturbed. The small finger is motionless. The proximal phalanges can not be flexed. Extension of the terminal phalanges of the last four fingers, and abduction and adduction of them are impossible. When the affection is of long standing, the characteristic picture of the "claw hand" is presented; in this case the first phalanges are strongly extended, the end phalanges completely flexed, and the interossei muscles are much atrophied.

Radial nerve lesion. The hand hangs in the condition of flexion, perfectly limp, and can not be extended. The fingers are flexed, the first phalanx can not be extended. The thumb is flexed and adducted and can neither be abducted nor extended. The extended forearm can not be supinated; in the condition of flexion, however, supination may occur by action of the biceps.

Phrenic nerve lesion produces paralysis of the diaphragm, with its characteristic modification of respiratory movements; that is, forcible superior thoracic respiration without the inspiratory protrusion of the epigastrium.

Peroneal nerve lesion produces the characteristic limp drooping of the foot, especially noticeable on walking. Dorsal flexion of the foot and toes, abduction of the foot, and elevation of its outer border are impossible. When of long duration there results a permanent position of the foot which necessitates the walking on the toe tips (*pes equinus*).

Tibial nerve lesion produces a loss of plantar flexion of the foot (the patients can not stand on their toe tips) as well as adduction of the foot and plantar flexion of the toes.

The diagnosis of lesions of the cranial or of the spinal nerves requires, irrespective of a knowledge of the anatomical relations, an investigation of the following data:

1. The *cause* must be determined, whether trauma, pressure,

exposure, or an infectious inflammation due to acute or chronic disease. 2. The *intensity* of the disease. This is recognized by the character of the electrical reactions (see p. 64). Three forms are recognized: *a. The mild form*, characterized by a normal electrical reaction of the paralyzed muscles. *b. Erb's middle form* by a partial reaction of degeneration. In these cases the irritability of the nerve diminishes without being extinguished, while the galvanic irritability of the muscles on direct application of the current to them is increased; the anode closure contraction is greater than the cathode closure contraction, $AnCc > CaCc$; all contractions are slow. *c. The intense form*, characterized by the complete reaction of degeneration, which manifests itself in a complete loss of faradic and galvanic irritability of the nerves, loss of faradic irritability of the muscles, in a quantitative and qualitative change in the galvanic irritability of the muscles; the contractions resulting from the application of an electrical stimulus are slow and wavelike. The anode closure contraction is equal to the cathode closure contraction, $AnCc = CaCc$.

Speech disturbance. — It is necessary to distinguish between speech disturbance due to functional inactivity of the muscles (*anarthria*) and defective speech production where the muscular apparatus is wholly intact (*aphasia*). Anarthria is the result of a lesion of the medulla oblongata (bulbar symptom).

In aphasia one must determine whether the patient has a true conception of the spoken word and is prevented only in transferring the proper concept into speech (*motor aphasia* or *ataxic aphasia*), or whether the conception of speech is so lost that he does not understand the sense of the word pronounced to him and is unable himself to produce word concepts (*sensory aphasia* or *amnesic aphasia*). The site of the lesion producing ataxic aphasia is in the convolution of Broca, the third left frontal convolution; that of sensory aphasia is in the first left temporal convolution.

Motor aphasia very frequently is associated with the inability to write, though the motor apparatus of the upper extremity be intact (*agraphia*), while sensory aphasia may be associated with an inability to read, though perfect vision may be present (*alexia*).

Ataxia is the inability to execute complex movements in a skilful manner when there is no disturbance of the motor apparatus; in other words, the inability to properly coördinate muscular action. It is most probably induced in this wise: after the destruction of the centripetal conduction paths, the movements are no longer controlled by the fine sensory impressions. Ataxia is the chief symptom of *tabes*; it occurs also in peripheral neuritis, is sometimes consecutive to alcoholism and diphtheria; in addition, it occurs in lesions of the cerebellum.

Ataxia of the hands is determined by having the patient, with his eyes closed, button his coat, by having him write, etc. Ataxia of the legs may be demonstrated by having the patient touch the knee of the left leg with the foot of the right, or to describe an arc of a circle, etc. Ataxia is more exaggerated when the patient is in the dark.

Romberg symptom is the swaying of the body while standing with closed eyes. It occurs chiefly in *tabes*, but may also be present in neurasthenia.

Gait. Slight disturbances of motion or of coördination of the legs may be distinctly recognized in the patient's walk; thus the spastic, the paretic, and the ataxic gait are spoken of.

MOTOR IRRITATIVE PHENOMENA

Spasm. We designate clonic, that is, interrupted, short contractions, when involving the entire body, as *convulsions*; long-continued contractions are known as *tonic spasms*; when spread over the greatest part of the skeletal muscular system, it is called *tetanus*.

Clonic and tonic spasms appear in:—

1. *Epilepsy.* In this affection they are first tonic, subse-

quently they become clonic, and are accompanied by total loss of consciousness, which persists throughout the attack, with dilated pupils, which will not react. The face is at first pale, then becomes cyanosed, and the tongue is often bitten.

2. *Eclampsia* of pregnant and parturient women, commonly accompanied by dropsy and albuminuria.

3. *Uræmia* appearing in the course of acute and chronic nephritis; in a few cases the nephritis may have been unperceived, and the first indication of the uræmic character of the spasms may only have been determined by the discovery of albumin in the urine.

4. As the result of a direct irritation of the cerebral motor centres, occasioned by *tumor, abscess, cysticercus*, etc.

5. In children as a consequence of the *increased reflex irritability* accompanying febrile diseases, dentition, indigestion, worms.

Pure tonic spasms are a feature of:—

1. *Tetany*. They are chiefly confined to the flexor muscles of both arms and both legs. The duration of the attacks may vary from minutes to hours, seldom days. As a rule, there are several attacks daily. The attack may be produced by pressure upon the larger arteries and nerve-trunks of the arm (*Trousseau phenomenon*). The temperature is normal. During the period of repose after the subsidence of an attack, the mechanical and electrical irritability of the peripheral nerves is increased. The *prognosis* is favorable.

2. *Tetanus*. The tonic spasms in this disease are confined chiefly to the muscles of the face, producing *trismus* and *risus sardonicus*; to the muscles of the back, resulting in *opisthotonus*, and to the abdominal muscles; those of the arms and legs are not as often affected. The continuous spasms are interrupted by attacks of jerking. The temperature of the body is increased, and hyperpyrexia is marked before death.

In cases of tetanus the diagnosis should also embrace a determination of the cause and the search for the existing wound through

which the tetanus-bacillus entered the system. The wound will often be found to be very slight, or even already healed. The prognosis may be more favorably affected at times when the foreign body (splinter, etc.) which caused the wound has been removed. It is also important to determine the period of incubation; the longer the duration of this, the better the prognosis.

Localized spasm in the course of distribution of certain nerves results partly as a reflex phenomenon, and partly as an individual disease in neuropathic subjects.

Localized tonic spasm occurs along the course of the trigeminus, in the muscles of mastication (trismus), in tetanus, meningitis, epilepsy, hysteria. In such cases artificial feeding may have to be used.

Painful spasm of the legs (cramp) is often noticed after strong muscular effort, in the hysterical and the alcoholic, and in isolated cases of gout and diabetes.

Rhythmical contractions which repeat themselves and which are partly continuous and partly occur at intervals are called *tic convulsif*. We recognize tic convulsif in the distribution of the facial (spasm of the muscles of mimicry), of the spinal accessory (tic of the sterno-cleido-mastoid), seldom, though, in the distribution of the spinal nerves (tic of the rectus abdominis, of the psoas).

Intention spasm or tonic spasm, occurring at the beginning of voluntary movements of the muscles, is the pathognomonic symptom of *Thomsen's disease (myotonia congenita)*. This disease is one of lifelong duration.

Every voluntary muscle which had been previously at rest becomes, when put into action, the seat of a mild tetanic contraction; the patient can not immediately relax the muscle at command, he is, therefore, unable to carry out coördinated movements; after long tedious motion the contraction becomes less intense. The electrical reaction is peculiarly altered, giving rise to the myotonic reaction of Erb.

Tremor in muscles at rest is the sign in nervous people of intense psychical excitement. *Persistent tremor* without any pathological significance occurs often in old people (*tremor senilis*), but it is characteristic of chronic *alcoholism* and of

Basedow's disease. Extensive tremulous movement is significant of *paralysis agitans*.

The tremor is slow (5 to 6 oscillations in a second) in the senile, in sclerosis and in *paralysis agitans*; it is quicker (10 to 12 oscillations) in alcoholics and in *Basedow's disease*.

Tremor in voluntary muscles which are put into exercise (*intention tremor*) is pathognomonic for multiple sclerosis; it disappears during sleep, however.

Tremor of the eyes (*nystagmus*) occurs in multiple sclerosis, in workmen who having been confined in the dark are suddenly exposed to light (*nystagmus of miners*), in hysteria, and in certain nervous affections of the eyes.

Choreic movements, involuntary uncoordinated movements, which embarrass voluntary movements and interrupt them, and which only cease during sleep, are pathognomonic of *chorea* (*St. Vitus's dance*), which is a functional neurosis. They occur very seldom in cerebral lesions.

Choreic movements also appear as an indication of an intense cerebral intoxication in infectious diseases, as in typhoid and miliary tuberculosis.

The following are not of essential significance:—

Athetoid movements. They are involuntary peculiar abductions and flexions of the fingers indicative of a special affection (*athetosis*) or are a symptom of certain central nervous affections (especially of cerebral palsies of children).

Imperative movements are noteworthy in lesions of the cerebellum; they occur as coördinated spasmodic movements (laughing spasms, screaming spasms, jumping spasms) in hysteria and epilepsy.

Cataleptic rigidity of the muscles is noticeable in hysteria, in the hypnotic state, also in meningitis, in certain psychoses (*melancholia attonita*). The limbs remain fixed immovably in any position in which they may be placed.

THE REFLEXES

It is necessary to distinguish between skin and mucous membrane reflex, tendon reflex, and reflex function, whose relations to each other may often be entirely different.

By *skin reflex* is meant the muscular contractions induced in a reflex manner by irritation of the sensory nerves of the skin.

This reflex may be elicited by tickling, pricking, stroking, by cold (touching with ice).

The usual examination comprises a test of :—

1. The *plantar reflex*, produced by irritating the sole of the foot, which is followed by a dorsal flexion of the foot and even of a flexion of the lower extremity on to the abdomen.

2. The *cremaster reflex*. Stroking the inner aspect of the thigh produces reflex movement of the testicle.

3. The *abdominal reflex*, produced by irritating the skin of the abdomen, which induces a contraction of the abdominal muscles of the same side.

4. *Gluteal, scapular, mamillary* reflexes are of slighter importance, and are often absent under normal conditions.

Diminution or *loss* of the skin reflex always results when the reflex path (centripetal nerve, anterior cornu of the cord, and motor nerve) is interrupted; therefore it is to be found in disease of the peripheral nerves and of the spinal cord.

Increase of skin reflex occurs in: 1. Cases of increased irritability of the parts concerned in the reflex are: *hyperæsthesia* of the skin, strychnia intoxication, certain neuroses. 2. Cases of restoration of certain interrupted processes, as in diseases of the brain and spinal cord.

The mucous membrane reflexes are: 1. *Conjunctival* and *corneal* reflexes, which manifest themselves in closure of the eyes when the conjunctiva and cornea are respectively touched. 2. *Retching*, by irritation of the pharynx. 3. *Sneezing*, when the nasal mucous membrane is irritated. 4. *Coughing*, on irritation of that of the larynx and respiratory passages.

Tendon reflex is the term applied to the contraction of a muscle which is induced by irritation of its tendon, periosteum, or fascia.

1. *Patellar reflex* (knee jerk, knee phenomenon). If the patellar tendon be struck by a percussion hammer¹ while the leg is flexed and hangs perfectly flaccid or limp, the leg is thrown into a forwards and backwards movement by the resulting contraction of the quadriceps extensor muscle. It is necessary in eliciting this phenomenon that the attention of the patient be withdrawn from his knee. For this purpose it is well to make use of *Jendrassik's* trick: to have the patient clasp his hands across his chest and to pull at them with all his strength; at the moment in which he is exerting the pull, the tendon should be struck unexpectedly.

2. *Achilles tendon reflex*. The Achilles tendon should be suddenly struck while the foot of the patient is slightly flexed; the gastrocnemius will thereupon distinctly contract. This reflex may be absent even in the normal person.

3. *Foot clonus* (foot phenomenon). The foot should be suddenly and forcibly flexed dorsally while the knee is slightly flexed; whereupon there follows an energetic tremor of the foot. It is very seldom found in health.

4. Tendon reflexes of the upper extremity appear very seldom in normal conditions.

The *loss* of tendon reflexes occurs whenever there is an interruption or break in the reflex arc. The reflex path is by way of the sensory nerves, the posterior columns of the cord, the anterior cornua, and the motor nerves. Hence the tendon reflexes are absent in all peripheral lesions, such as multiple neuritis, diphtheritic and alcoholic neuritis, and traumatic lesion, in the degenerations of the posterior columns, as in tabes, and in the diseases of the gray substance of the lumbar cord, as in poliomyelitis.

Recently the loss of tendon reflex has been demonstrated in cachectic conditions as they occur in pernicious anæmia, and in the grave form of diabetes.

Exaggeration of reflexes occurs whenever the reflex inhibitory centres are diseased. These centres are probably situated in the brain, and send out their impulses along paths which proceed through the pyramidal tracts of the

¹ The finger tips will answer the same purpose. — THE TRANSLATORS.

cord. Therefore exaggeration of the reflexes will be found in cerebral paralysis, as well as in chronic myelitis; that is, in that form which gives rise to spastic paralysis.

Reflex functions: 1. Pupillary reaction to light and to accommodation.

The pupillary sphincter muscle is supplied by the motor oculi nerve and the pupillary dilator by the sympathetic. Irritation of the motor oculi results in contraction of the pupil; irritation of the sympathetic produces pupillary dilatation. To these conditions the respective names of myosis and mydriasis are given. Paralysis of the motor oculi produces dilatation, that of the sympathetic, contraction of the pupil. The centre is said to be placed in the inferior cervical segment of the cord (*centrum cilio-spinale*). (Compare p. 47.)

The pupillary reaction may be absent in various cerebral affections. *Reflex iridoplegia* is of very great importance as a diagnostic sign of tabes dorsalis. It shows itself as follows: the pupil contracts on accommodation, but not to light. At the same time it very frequently occurs in tabes that the pupils are extremely small (*myosis spinalis*) and unequal in size. Reflex iridoplegia further is an early symptom of parietic dementia.

2. Disturbances of defecation and of micturition, as well as of the sexual reflex (vesical tenesmus, incontinence, constipation, but seldom incontinence of fæces) are pathognomonic of disease of the lumbar cord, especially of tabes and diffuse myelitis. Impotence often appears early in locomotor ataxia.

Impotence is also a symptom of diabetes and Bright's disease, as well as of conditions of irritable weakness or exhaustion in neurasthenics.

Of less diagnostic importance is the direct mechanical irritability of the muscles, which, as a rule, are well preserved.

Paradoxical contraction of Westphal, which occurs among other diseases in multiple sclerosis, paralysis agitans, has been up to this time of little value. It consists in the fact that when the foot is

passively flexed dorsally, it will remain for some minutes in this position, even after being left alone, and the tendon of the tibialis anticus muscle becomes prominently visible.

THE DISTURBANCES OF SENSATION

The diagnosis of every disease of the nervous system can only be completed after a thorough test of sensation. Disturbances of sensation are pathognomonic of many diseases of the brain and spinal cord; for instance, lesion of the internal capsule, tabes, syringomyelia, neuritis. In many other cases the extent and intensity of the diseased process can only be determined by an examination of the sensation.

We designate as *anæsthesia* the loss or diminution of sensation; *hyperæsthesia* the increase of the same so that very weak irritations are perceived as strong ones. *Paræsthesias* are abnormal sensory perceptions, such as itching, crawling, formication, the sensation of being stroked with fur, etc. *Neuralgias* are pains, chiefly paroxysmal, in the course of certain nerves. *Pressure points* are those points where the nerve appears directly beneath the skin or on the bone which are especially painful on pressure.

The neuralgias are special affections, as, for instance, trigeminus neuralgia, supraorbital neuralgia, sciatica.

There are special forms of neuralgia which recur paroxysmally at a definite period of the day and which are occasioned by malarial infection (malarial neuralgia, latent intermittent); these forms succumb to large doses of quinine.

A complete test of sensation must regard the various *qualities of sensation* of which each may be tested in various ways (partial anæsthesia).

We should test tactile sense, pain sense, space sense, pressure sense, dynamic sense, muscular and articulation sense, temperature sense, and electro-cutaneous sense.

These tests should be made at various periods, as the undivided attention of the patient is necessary.

1. *Tactile sense.* The various regions of the skin should be touched lightly with a brush, the eyes of the patient should be closed, and he should be directed to say "yes" at each touch. In some cases, especially in tabes, the answer of the patient may come some time after the touch (delayed conduction).

Hyperæsthesia in the extremities occurs among other symptoms as a sign of neuritis; it occurs also in the prodromal stage of some acute infectious diseases, as in typhoid, for instance, and during the entire course of meningitis.

2. *Pain sense* is tested by pricking with a pin, by pinching, and with strong electric currents. *Analgesia* is the lack of sensation (irrespective of the ordinary sense of touch) to the usual painful irritants, such as the prick of a pin; it occurs in hysteria, tabes, and peripheral neuritis. In some individual cases a *delayed painful sensation* may be demonstrated; at times tactile sensation and pain sensation are felt one after the other (*double sensation*).

3. *Space sense or sense of locality.* The patient should be touched and he should immediately indicate the spot which was touched or place his finger upon it. The examination of the *tactile distance* belongs under this head. It is determined by the repeated application of a compass with widely opened legs, which are brought closer and closer together until the smallest distance apart in which they are felt as two tactile impressions is registered. This distance in the normal individual is for the

Tip of tongue, 1 mm.

Tip of finger, 2 mm.

Mucous surface of lip, 3 mm.

Dorsal surface of 1st and 2d phalanges and inner surface of finger, 6 mm.

Tip of nose, 7 mm.

Thenar and hypothenar eminences, 8 mm.

Chin, 9 mm.

Tip of large toe, cheeks, and eyelids, 12 mm.

Heel, 22 mm.

Dorsum of hand, 30 mm.

Throat, 35 mm.

Forearm, leg, and dorsum of foot, 40 mm.

Back, 60 to 80 mm.

Arm and thigh, 80 mm.

4. *Pressure sense.* Considerable disturbances of this sense are recognized by applying various strong degrees of pressure with the

finger, slighter disturbances by applying various heavy weights upon the extremities, which ought to be supported upon a firm base. The patient should mention the differences between the weights. A healthy person can recognize differences of one-tenth of the original weight; the smallest difference appreciable varies from 0.005 to 0.5 g.

5. *Dynamic sense* is tested by lifting in the hand various heavy weights which have been wrapped in a cloth and estimating their weights. In the normal person the dynamic sense is finer than the pressure sense.

6. *Articulation sense and muscle sense.* By means of these senses the position of each limb is determined with closed eyes. It is tested, for example, by flexing the fingers of the patient to smaller and larger degrees and fixing them, when the patient is to recognize the various degrees of excursions or flexions with closed eyes; or the leg is to be abducted and fixed in the position in which the slightest degree of abduction may be recognized by him. Disturbances of the articulation sense may be demonstrated in all cases where there is an interruption of the sensory conduction tracts, particularly in tabes and neuritis, mostly before the gross ataxic disturbances become distinct.

7. *Temperature sense.* The skin is touched with dry test tubes or metal cylinders, some of which have been filled with cold and some with warm water. In health, differences of $\frac{1}{2}^{\circ}$ to 1° C. may be detected between 25° and 35° C. marks of the thermometer. *Perverse temperature sense* is the term given to the disturbance of sense in the patient who recognizes a sensation of heat when touched with ice. Another more recent test of the temperature sense consists in comparing, in the various regions of the skin, the absolute temperature sensibility which under normal conditions show constant fixed local differences (*Goldscheider*).

8. *Electro-cutaneous sensibility.* A faradic current is applied with a wire brush and the figure is read from the instrument after the secondary coil is pulled out to the extent at which the current is first felt.

EXAMINATION OF ELECTRICAL IRRITABILITY

The examination of the electrical irritability of paralyzed muscles and nerves leads to important conclusions concerning the location of the disease, but especially to a more definite prognosis of the duration of the disease.

Method and normal relations.—An electrical examination is conducted with the faradic (induction) and with the galvanic (constant) current. The indifferent electrode is placed upon the sternum, the other upon the muscle or nerve to be examined. The excitation of the muscle by way of the nerve is called *indirect irritation*; that resulting from the direct application of the electrode to the muscle itself, *direct irritation*. The examining electrode for determining the irritability of each muscle or nerve is placed upon certain fixed points which have been determined empirically and may be seen in Figs. 18 to 22.

When the faradic current is applied by direct and indirect irritation, a distinct contraction results. The distance in millimetres to which the secondary coil is moved to produce the slightest noticeable contraction is then read. It is denoted in all German books as R.A.—mm.

In the *galvanic examination* the electrode used is converted by means of a *pole changer* to first the *negative (cathode, zinc) pole*, then to the *positive (anode, carbon, or copper) pole*. On gradual increase of the strength of the current the first weak contraction of the muscle appears, providing the current is closed in such a manner that the examining electrode is made the cathode pole (CaCc). The amount of current used (determined by the number of cells used or by the absolute galvanometer) is then registered. With this amount of current, reaction will not yet appear on cathodal opening, anodal closure, or anodal opening. By increasing the strength of the current a gradual appearance of contraction will appear on anodal opening (AnOc) and on anodal closure (AnCc); only with a much stronger current than this will cathodal opening contraction appear, and then the cathodal closure contraction will already have been tetanic. The order in which contractions of a *normal* muscle takes place by increasing strength of current and *indirect* irritation is, hence, as follows:¹ 1. CaCC; 2. AnOC; 3. AnCC; 4. CaCTe; 5. CaOC. Contractions are short and lightning-like.

¹ The following abbreviations are in use: Ca = cathode; An = anode; C = closure; O = opening; c = slight contraction; C = strong contraction; Te = tetanus.

Quantitative change of electrical irritability. In different diseases the electrical irritability of the nerves and muscles is simply increased or diminished without there being any change in the order or in the quality of the contractions.

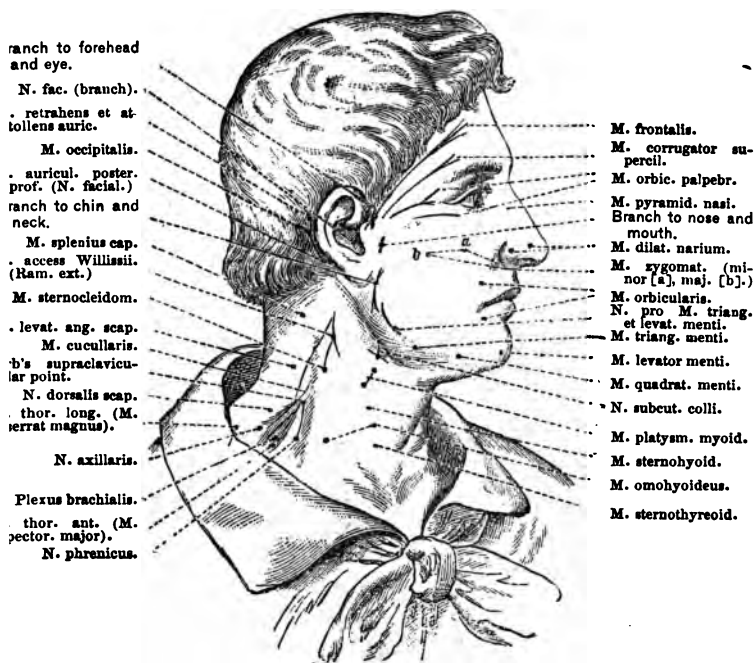


FIG. 18.

In order to compare the results of examination of a unilateral paralysis, the corresponding nerves and muscles of the unaffected side should be tested as well. In bilateral paralysis and in general disease of the nerves a comparison is made with the electrical irritability of certain nerves as it occurs in the normal person, the nerves used being the frontal, accessory, ulnar, peroneal nerves.

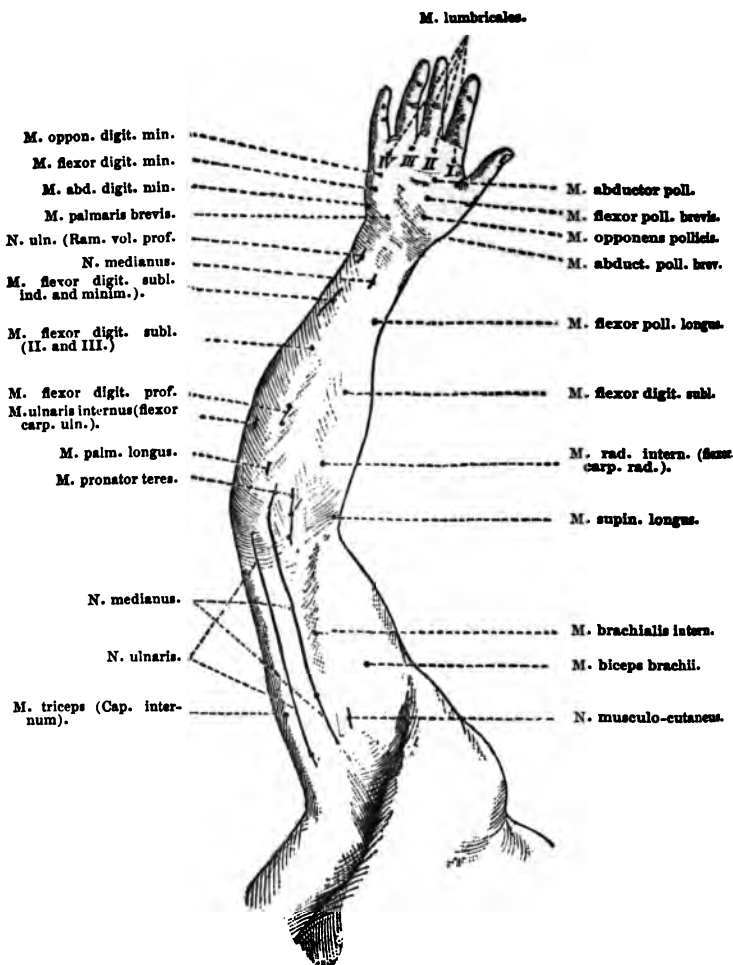


FIG. 19.

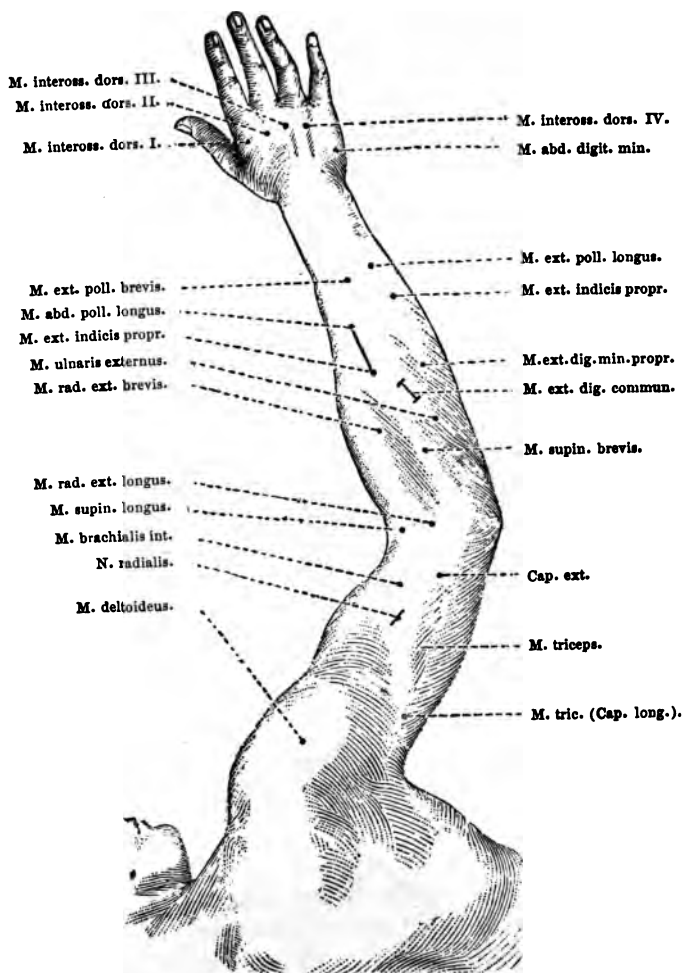


FIG. 20.

Electrical irritability is *simply increased* especially in tetany, and in cases of recent peripheral lesions.

Electrical irritability is *simply diminished* in all paralysis of long standing which leads to muscular atrophy, as in cerebral hemorrhage, bulbar lesion, cord lesion where the trophic centres are intact.

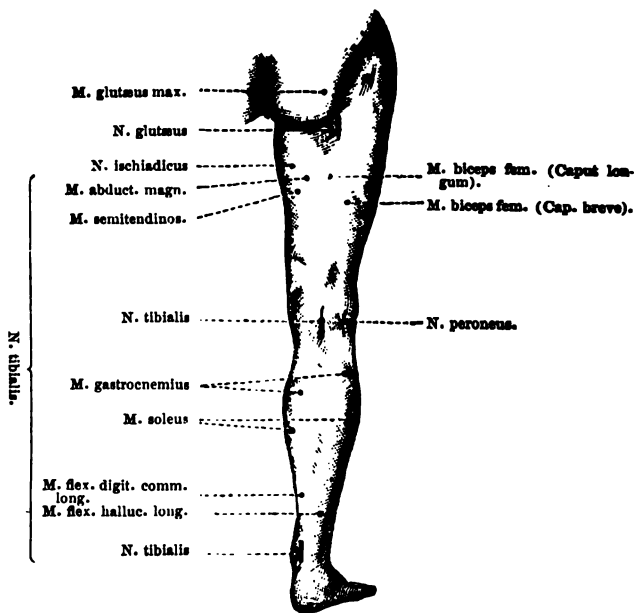


FIG. 21.

Qualitative change of the electrical irritability: Degeneration reaction (R.D.). Qualitative change of the electrical irritability is an infallible sign of paralysis of the nerve *peripherally*; that is, in cranial nerves the gray nuclei, in spinal nerves the ganglia of the anterior cornua are themselves diseased or the disease lies *peripherally* to these trophic centres. In these cases the *faradic and galvanic*

irritability of the nerves diminish more and more a short time after the beginning of the paralysis. In 8 to 10 days the irritability is absolutely lost; no contractions are to be obtained even with strong currents. The direct faradic irritability of the muscles is also abolished.

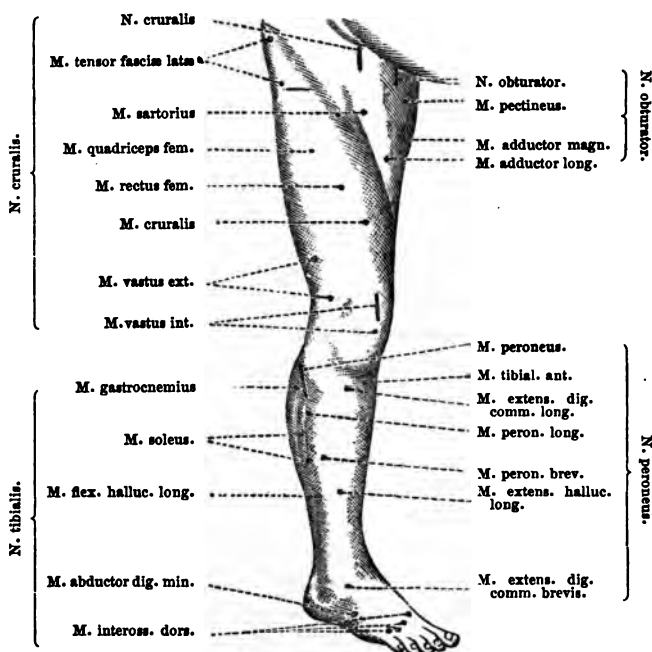


FIG. 22.

On the other hand, the direct *galvanic irritability of the muscles* is appreciably increased from the second week; contractions result even with the weakest currents. The contractions are *slow, long continued*, and vermicular. The AnCC follows upon the CaCC with the same strength of current, and is often stronger than the CaCC; frequently

CaOC appears sooner than AnOC with less strong currents (complete degeneration reaction).

In severe, more especially in incurable, cases the galvanic muscular irritability diminishes after 4 to 8 weeks' duration of the disease, until it completely disappears. In curable cases the conditions of electrical irritability become gradually normal; in such cases the power of voluntary movement of the muscle recovers, however, often much sooner than the electrical irritability.

Partial degeneration reaction, which permits of a diagnosis of paralysis of lighter intensity, consists in this, that faradic and galvanic irritability of the nerves and faradic irritability of the muscles are only slightly lowered, whereas the distinguishing characteristic changes on direct galvanic irritation of the muscle are entirely equal to each other (see p. 49).

The trophic condition of the paralyzed muscles is in harmony with the electrical relation; disease of the ganglia of the anterior cornua, as well as of the nerves peripheral to these, leads to *degenerative atrophy*; disease central to the trophic centres leads only gradually to slight diminution in bulk of the paralyzed muscles.

Degeneration reaction occurs in peripheral lesions of the motor nerves (traumatic and rheumatic, diphtheritic, toxic paralysis, multiple neuritis, alcoholic neuritis), in disease of the gray anterior cornua, and the gray nuclei of the medulla (atrophic palsies of children, poliomyelitis).

Degeneration reaction is absent in all cerebral and in all spinal paralysees whose origin is cephalad to the trophic centres.

Degeneration reaction is of prognostic importance in this, that either irreparable atrophy of the affected muscles will ensue or that recovery can only at best begin to take place in 2 or 3 months. Absence of degeneration reaction is a certain diagnostic sign that no gross anatomical changes have

taken place; it is of prognostic value in that it denotes that recovery will ensue in a short time, often in 3 to 4 weeks.

Partial degeneration reaction indicates that the muscles have suffered serious anatomical changes, whereas the nerves have been unaffected; it admits, therefore, as far as time is concerned, of a better prognosis than complete degeneration reaction.

SYMPTOMS OF SOME OF THE DISEASES OF THE NERVOUS SYSTEM

Cerebral palsies have been spoken of sufficiently in the beginning of the chapter (see p. 43 *et seq.*).

Cerebral abscess is diagnosed by: (1) the etiology (trauma, otitis, embolism); (2) the general symptoms (headache, vertigo, vomiting), varying in intensity; (3) the irregular remissions of the fever; (4) the absence of choked disc; (5) the focal symptoms, which vary according to the site of the abscess, although they may be entirely absent.

Tubercular meningitis is diagnosed when: (1) other organs are demonstrated to be the seat of a tuberculous process; (2) the fever is of irregular remittent type; (3) the pulse is slow and irregular; (4) the stupor is severe and associated with delirium; (5) the neck is rigid, there is vomiting, and the abdomen is retracted; (6) finally tubercle bacilli in the cerebro-spinal fluid obtained by lumbar puncture are found.

Cerebral syphilis may be diagnosed from the following, if proof of previous syphilis can be obtained: there are often intense prodromal headaches, epileptiform convulsions, and apoplectiform attacks, various paralyses. The general signs and the focal symptoms are not at all characteristic, for the same may be present in tumor, hemorrhage, etc. *In all doubtful cases mercurial inunctions should be given, which, if remedially effective, would determine the diagnosis.*

Tumors of the brain. — A diagnosis may be made from the following data: (1) The general symptoms, such as head-

ache, vertigo, vomiting, convulsions, psychic weakness, are ushered in gradually, and increase slowly in intensity; (2) ophthalmoscopic examination reveals choked disc, though an absence of this condition does not disprove the presence of a tumor; (3) there are focal symptoms which vary, of course, according to the region of the brain, which is the seat of the growth.

Progressive bulbar paralysis is accompanied by disturbance of speech (anarthria), atrophy of the tongue, of the lips, and of the muscles of mimicry supplied by the facial nerve. Owing to this paralysis, the face appears mask-like. There is difficulty in swallowing, arising from the atrophy of the pharyngeal muscles. Speech is weak, and its tone unvarying (monotone), owing to atrophy of the laryngeal muscles; coughing becomes impossible, and finally respiratory paralysis supervenes.

Degeneration of the combined pyramidal tracts (manifested by muscular atrophy and increased tendon reflexes in the upper and spastic paralysis in the lower extremities) is associated with degeneration of the nuclei of the medulla. This condition has been recognized by a few authors as a distinct and special disease, which they call *amyotrophic lateral sclerosis*.

In some cases bulbar symptoms are associated with *progressive spinal muscular atrophy* (see below), which consists of a degeneration of the anterior cornua and the anterior nerve-roots.

All three diseases present a degeneration of the same motor conducting apparatus, but in different locations, so that a diagnostic separation in many cases appears artificial.

Myelitis. — The symptoms vary according to the position of the process in the cord.

Myelitis cervicalis is characterized by paraplegia of the legs, paralysis and sensory disturbances in both arms. The tendon reflexes are increased, and spastic phenomena are present. The bladder and rectum are affected.

Myelitis dorsalis presents essentially the same symptoms, but the arms are wholly unaffected.

Myelitis lumbalis. Paraplegia of both legs, but the upper extremities are unaffected. The bladder and the rectum are involved; the skin and tendon reflexes are diminished or entirely absent.

As a special form of chronic myelitis, there have been described: (1) the so-called *spastic spinal paralysis* (*Erb, Charcot*), characterized by spastic symptoms in the extremities, increased reflexes with the preservation of the bladder and sexual functions, and said to depend wholly on lesion of the pyramidal tracts; and (2) *progressive spinal muscular atrophy*, which gradually leads to degenerative atrophy of the muscles of the arm and shoulder, and which is due to a lesion of the gray anterior cornua and anterior roots.

The description of *poliomyelitis* (lesion of the gray substance of the cord) belongs to this category. It is often confounded with multiple neuritis, and its diagnosis is made with great difficulty. Poliomyelitis can only be diagnosed with certainty when it occurs in children, where it is known as the *essential (spinal) palsies of children*. Here the age, the acute onset, the subsequent flaccid paralysis, accompanied with atrophy and the presence of degeneration reaction, the absence of reflexes, and the preservation of sensation, will establish the diagnosis.

Tabes dorsalis is diagnosed by the iridoplegia, the loss of tendon reflexes, lightning-like pains in the legs, the ataxia, the Romberg symptom, analgesia, at times anæsthesia, delayed conduction, and paræsthesia. In some cases there are periodical disturbances of the abdominal viscera, as, for instance, frequent painful attacks of vomiting (*crises gastriques*). The presence of two characteristic symptoms (for instance, iridoplegia and failure of the reflexes, or iridoplegia and *crises gastriques*) will render the diagnosis certain. It is distinguished by three stages: the neuralgic, ataxic, and paraplegic.

Multiple sclerosis of the brain and spinal cord. — In typical cases there is intention tremor, scanning speech, nystagmus, spastic paretic gait, increased tendon reflexes, foot clonus, and gradually developing psychological weakness. Only seldom do we find all signs present in a single case.

CHAPTER IV

DIAGNOSIS OF THE DISEASES OF THE DIGESTIVE SYSTEM

Lips.—The condition of the nutrition (see p. 4) of the patient and the state of the blood (see p. 7) are determined by the color of the lips. Dryness of the lips, and crusts thereon, indicate a febrile disease. In typhoid, the brown, rusty color of the lips (*fuligo*) is characteristic.

Teeth.—A healthy person has moist teeth. Dry teeth covered with sordes indicate neglect and fever. A good nurse should see that the lips, mouth, and teeth of every comatose patient are kept moist and clean.

A good condition of the teeth insures good mastication. When many teeth are wanting, the food is swallowed imperfectly masticated; very often their absence serves as a guide to the diagnosis of chronic gastritis.

The *age* of children may readily be determined by the number of their teeth, and especially at the time of their appearance. A knowledge of the period of the eruption of the teeth is necessary for the diagnosis of many diseases of children.

The milk teeth are twenty (20) in number; on each side above and below, two (2) incisors, one (1) canine, and two (2) molars. They appear from the 3d month to the 3d year of life, usually in the following order: the lower central incisors from the 3d to the 10th month (average 7th month), the upper central incisors from the 9th to the 16th month, the superior lateral incisors from the 10th to the 16th month, the lower lateral from the 13th to the 17th month. The first four molars from the 16th to the 21st month, the four canines from the 16th to the 25th month, and the four second molars from the 23d to the 36th month, as an average, however, between the 24th and 30th months.

The change to the permanent teeth begins in the 7th year of life and proceeds, as a rule, in the same order as does first dentition. The third molars (wisdom teeth) appear from the 18th to the 30th years of life. The permanent teeth are thirty-two (32) in number: two (2) incisors, one (1) canine, two (2) bicuspid, three (3) molars on each side above and below.

Tongue. — An inspection of the tongue is sanctioned by old custom and should begin the examination of the patient. The tongue of the healthy is bright red and moist, and has no tremor when extruded.

In febrile diseases, before the period when a competent nurse takes charge, the tongue is, as a rule, dry, fissured, and covered with discolored fur. Its appearance in typhoid and in scarlet fever is characteristic; it is covered with a white fur, sometimes brown, and with a red stripe on either side in typhoid; it has the strawberry appearance in scarlet fever (strawberry tongue).

In non-febrile conditions the tongue should be examined to see if it be bright red or coated.

A normal condition of the tongue in these respects would in most cases exclude disease of the stomach. A coated tongue often indicates a catarrhal condition of the gastric mucous membrane. Still these conditions are not regularly united, so that the diagnostic deductions from the coated tongue must be drawn with care. It should be borne in mind that ulcer of the stomach (*ulcus ventriculi*) and hydrochloric hyperacidity are usually unaccompanied by a coated tongue.

Glossitis or inflammation of the tongue, attended by swelling of and intense pain in this organ, is an infrequent but severe infectious disease which requires chiefly surgical treatment.

Mouth. — Inflammation of the mucous membrane of the mouth (*stomatitis*) is recognized by swelling and œdema of the mucous membrane which is exceedingly painful. It is occasioned chiefly from neglect and often from the use of

mercurials. It is relieved by rinsing the mouth with potassium chlorate.

A mild form of stomatitis not infrequently develops after a generous consumption of grapes such as is ordered in "a grape cure."

A reddish border at the junction of the gums with the teeth occurs often and has no diagnostic significance; a grayish or bluish border is produced by plumbic sulphide and indicates plumbism (lead intoxication); it is the so-called blue line of the gums.

Soor or thrush consists of small grayish-white membranous exudations upon the buccal mucous membrane which occur in weakly children often after neglect and uncleanness, and develop in the adult chiefly toward the end of some severe disease (phthisis, sepsis, etc.). (See Chap. XII.) The cause of this disease is a fungus (*Saccharomyces* or *Oidium albicans*) which grows in acid media in buds and in alkaline media in threads. In the mouth it grows in threads and round conidia.

Pharynx and tonsils.—(See Chap. V., Diagnosis of the diseases of the upper air-passages.)

Saliva is an alkaline glandular secretion containing mucin, whose active principle is *diastase*, which changes starch into sugar. In stomatitis and in a few diseases, such as diabetes, the saliva becomes acid. In hyperacidity of the stomach the diastatic activity is often reduced. Still, thus far, the examination of the saliva has not attained any diagnostic importance.

Saliva is recognized by the blood-red color obtained by adding an acid and precipitating with ferric chloride. This reaction depends upon the presence in saliva of rhodanpotassium (CNSK).

ŒSOPHAGUS

Of the diseases of the œsophagus *stricture* of this organ has a special diagnostic importance. It is determined by the complaint of the patient that the bolus of food sticks in

his throat or before it reaches his stomach, and that it is often regurgitated and tastes very badly.

About the site of the stricture a dilatation is produced by the pressure of the accumulated food, in which decomposition and fermentation of the food may take place. The retained remnants of food are regurgitated after a while by the retching produced from the irritation. We can recognize that these remnants never reached the stomach by the fact that they are alkaline in reaction, owing to the admixture of saliva, and where milk has been taken by the fact that it is not coagulated.

A special form of constriction is the *diverticular* (a dilatation of the mucous membrane *without a primary stenosis*). We distinguish between a *pressure* diverticulum and a *traction* diverticulum. The former is always situated in the extra thoracic part of the œsophagus, reaches a considerable size, varying between 5 and 10 cm., so that when distended it may be seen and felt on the outside of the neck as a distinct *tumor*. This form of diverticulum may shut off the way for the entrance of nutriment and thus cause death from inanition. It is produced by pressure of foreign bodies, swallowing too large masses of food, and by traumatism. The *traction diverticula* occur in the lower half of the œsophagus, and have a minimal size of 4 to 8 mm. They are scarcely ever the object of medical diagnosis. They are produced by the traction resulting from cicatricial formation; in rare cases they have led to pulmonary gangrene and death by perforating into the bronchi.

The diagnosis of œsophageal stenosis is made with certainty by introducing a moderately hard œsophageal sound of ordinary calibre. Before this is done it is absolutely necessary to examine in each case whether there is an aortic aneurysm present; should such be the fact, no attempts at introducing the instrument should be made. Recently success has been attained in obtaining a view of the interior of the œsophageal tube by means of direct œsophagoscopy (*Rosenheim*); but this method of examination is too difficult to admit of general use.

It is necessary to diagnose: 1. *The seat of the constriction*, as the surgical possibility of relief will be thereby determined. To determine this point, mark the place on the

œsophageal sound where the teeth touch it after the constriction has been reached, and after the instrument has been removed measure the length of the part which has been included between these two points.

The distance between the upper incisor teeth and the cardia of the stomach measures in adults, as a rule, 40 cm., between the incisors and the beginning of the œsophagus the distance is 15 cm., and from the incisors to the point where the œsophagus crosses the bronchus is 23 cm.

2. *The cause of the constriction*, as from this directly depends the prognosis and treatment. The most frequent cause of constriction is *carcinoma*. This is to be diagnosed in old people, especially if they emaciate rapidly and have a cachectic appearance, if there be no special reasons against it.

In the young the following causes may occasion the stenosis: 1. *Cicatricial formation*, (a) the result of an extensive burn by acids or alkalies, which may have been swallowed accidentally or with suicidal intent, (b) the result of an ulcer of the cardiac orifice of the stomach or of the œsophagus, which has given symptoms for years of a round ulcer. 2. *Esophagitis*, which arises from swallowing a foreign body or from the extension of inflammation from a neighboring pus formation. 3. *Tumor of the mediastinum or aortic aneurysm*, which may be determined by the physical signs. 4. *Cheesy bronchial glands* at the hilus of the lungs, which may be presumed to exist when tuberculosis of the lungs accompanies the stenosis. 5. *Syphilis*, which seldom leads to œsophageal stenosis, and which can only be assumed when syphilis is shown to be present and when every other etiological factor has been excluded. 6. *Hysteria* in the young should cause one to think of the possibility of a nervous spasm of the cardia.

The diagnosis is only completed when the *permeability* and the *calibre* of the stricture are determined; one should try to pass the stenosis with carefully repeated applications of sounds of varying calibre. The proof of the permeability of the stricture is not always to be found in the descent of the sound, for that may be occasioned by the sound bending upon itself in a diverticulum, but only upon hearing the *deglutitory murmur* when the patient is asked to swallow.

This sound is heard on auscultation: 1. Posteriorly, on the left side next to the vertebral column at the level of the 6th dorsal vertebra; it is a short, dull murmur, which may be heard immediately after the act of swallowing. In stenosis it is exceedingly weak, and in closure of the cesophagus it is entirely absent. 2. Anteriorly, over the arch of the ribs on the left side next to the xiphoid process. Often, in addition to the deglutitory (primary) murmur, another (secondary) murmur, occurring from 3 to 5 seconds later, may be heard, or even a third; these secondary sounds are most probably produced by regurgitating bubbles of air. In stenosis these secondary murmurs are heard from 5 to 12 seconds later.

Above a stenosis and over diverticula, loud sounds, at times lasting many minutes, may be heard on auscultation; they arise from the motion transmitted by muscular action to the fluids retained in the diverticula.

THE DIAGNOSIS OF THE DISEASES OF THE STOMACH

As far as the history of the cases is concerned, the following points are to be regarded:—

Hereditary influences have seldom an important bearing, except for carcinoma and neurasthenia. The patient's mode of life, however, is of greatest importance, whether he has been exposed to injurious occupation such as sedentary work, troubles and worries, psychical emotions, lead intoxication, etc., whether he was guilty of *excesses in diet*, or bolting of food, or of chewing his food imperfectly, or of taking his food too hot, whether he was an alcoholic or indulged excessively in tobacco. An essential point to be elicited is whether he became emaciated rapidly.

The next step is to investigate accurately the complaints of the patient concerning the signs of dyspepsia, to leave nothing omitted, and to follow with an inquiry concerning any casual former troubles in the other organs (lungs, heart, and kidneys).

The complaints of all patients with gastric affections, and those by which the attention of the physician is directed to the stomach, refer to the general so-called dyspeptic symptoms, viz. loss of appetite, eructations, heart-burn, pressure and fulness in the region of the stomach, pain in that organ, depression. None of these symptoms in itself is character-

istic enough to justify a definite diagnosis of the disease of the stomach. It is of special diagnostic value that the dyspeptic symptoms occur also in the course of diseases of other organs, as, for instance, in the inception and progress of pulmonary consumption (phthisical dyspepsia), in heart diseases during the stage of compensatory disturbance, in nephritis, in attacks of gout, in diabetes.

The appetite is an important indication of health. *Loss of appetite* is a sign of a general pathological disturbance, though no special deductions may be drawn from it. Most of the febrile and chronic diseases are accompanied by it. The special forms of gastric diseases which begin with a loss of appetite are gastritis and carcinoma. Gastric ulcer and hyperacidity, on the other hand, are usually associated with a good appetite. Still numerous exceptions occur. Excessively increased appetite (*bulimia*) as well as its perversion, pathological longings, are chiefly the signs of gastric neuroses, although they sometimes occur in other affections.

It was the custom to regard the condition of the appetite as an infallible sign of either good or bad digestive power; this is, however, only conditionally true. In many cases even a poor appetite may be associated with good digestive power.

Eruclation. — This symptom indicates the presence of fermentation in the stomach; it has no differential diagnostic significance.

Frequent loud eruclations, spasmodic in character, occur in neurasthenics as a result of swallowing air; the gas discharged is without smell and tasteless; chronic pharyngitis is often present in these cases.

Pyrosis, heart-burn, acid burning in the cesophagus. This is almost always a sign of an increased acidity of the stomach. Still no diagnostic deduction should be made from its presence, because it may just as well indicate an excess of hydrochloric acid (anorganic hyperacidity), a severe fermentation which appears first in cases of deficiency of hydrochloric acid (anorganic anacidity), and which leads to the formation of acetic acid, lactic acid, and butyric acid (anorganic hyperacidity). These conditions of anorganic and organic hyperacidity have different prognostic values, and demand, in fact, different forms of diet and treatment, notwithstanding both may present the same symptom of pyrosis.

Feeling of pressure and fulness in the stomach. This symptom is present in many gastric affections, is frequently purely a neurotic symptom, and is often the result of over-exertion.

Pain in the stomach. This symptom is only to be utilized with the greatest care in making a differential diagnosis. It frequently is present in ulcer, and, on the other hand, in gastric neuroses and catarrhs as well as in carcinoma. Only those pains which are confined to a *circumscribed spot* and which are always felt in that spot are of value in concluding that there is a gastric ulcer. (Localized painful points are found also in the neuroses; see also p. 81.)

VOMITING

Vomiting results when the centre situated in the medulla oblongata is irritated and at the same time the abdominal muscles and the diaphragm are contracted, the pylorus is closed, the cardia is open, and when probably antiperistaltic action is set up in the stomach. The irritation of the vomiting centre is induced either directly by the blood state, as appears when emetics are taken, by swallowing caustic materials such as acids and alkalies, or by poisons such as chloroform and morphia, or by toxic disease-products as in chronic *nephritis*, *uræmia*, and *cholera*, or by reflex irritation. Reflex vomiting may arise from various organs; from the *brain* in meningitis and tumor cerebri, from the *peritoneum* in peritonitis and appendicitis, from the *kidneys* in renal colic and pyelitis, from the *bladder* in stone, from the *genital organs* in endometritis and *pregnancy*, or from the *stomach* as in several diseases of that organ. The tendency to vomit in *heart disease* is chiefly induced by congestion gastritis, though often acute cardiac dilatation (for instance, induced by over-exertion) as well as cardiac failure are accompanied by it.

Vomiting may also result from psychic influences, especially from disgust. Many persons are nauseated from fright and fear.

Repeated vomiting is a sign of various visceral disorders. It is pathognomonic for meningitis, peritonitis, and uræmia. In Bright's disease it is an evil omen, as it is early indication of ensuing uræmia. Its frequency is for a great part a determining prognostic feature of meningitis. *Pregnancy* is often first diagnosticated by the presence of persistent vomiting, especially when the vomiting occurs with an empty stomach; a special prognostic and very serious symptom is

offered by the intractable vomiting of pregnancy, which often may be the indication for the artificial production of premature labor.

Repeated vomiting in *febrile disorders* demands special diagnostic attention. There may be *prodromal vomiting*, as occurs in scarlet fever and erysipelas. It may serve as an *essential symptom of disease*, as in meningitis and peritonitis. It may be caused by *drugs*, e.g., antipyrin, or by *defective nutritive materials*. Its most severe form is associated with "irritative exhaustion" and is then often accompanied by *singultus*, which are chiefly features of the remission of the disease, or of a beginning convalescence.

Periodical recurrent attacks of frequent vomiting which alternate with periods of freedom therefrom have a special diagnostic importance: they are the so-called *gastric crises*, and are associated with distressing, often unbearable nausea. They may last for days and frequently lead to inanition. They appear in diseases of the spinal cord, especially in *tabes dorsalis*. Not infrequently this characteristic group of symptoms may be the first sign which calls attention to the existing tabes which may have previously been overlooked. Still periodical recurrent vomiting may be a sign of a neurasthenic gastric disorder without having a central origin, but in each of the latter cases it is wise to entertain a suspicion of its being of central origin.

From vomiting *alone* no diagnosis of disease of the stomach should be made; there should be a further examination to determine whether any of the following described signs are present.

Vomiting in diseases of the stomach. Repeated vomiting occurs in so many gastric disorders (e.g., ulcer of the stomach, gastritis, dilatation of the stomach, carcinoma, gastric neurosis) that from this sign alone no diagnosis is possible.

Composition of the vomited material. The vomited matter is composed chiefly of particles of food which have been much changed by the gastric juice and by fermentation in the stomach (these

changes produce from the carbohydrates, lactic acid, butyric acid, and acetic acid; from the fats, free fatty acids; from the albuminoids, leucin, and tyrosin in addition to the peptones): saliva (especially present in matutinal vomiting); mucus (in gastritis, but not diagnostic); bile (with no diagnostic significance, yet it indicates always that the pylorus is permeable); urea (in uræmia; concerning its demonstration, see Chap. VIII.).

Microscopic examination of the vomited material. Under the microscope we find particles of food (striped muscular fibres, fat globules, starch cells, and vegetable fibres), pavement epithelium, which comes from the mouth and œsophagus, leucocytes, various forms of bacilli and cocci, fungus, and sarcina. An abundance of sarcina indicates a considerable fermentation in a dilated stomach, though it gives no indication as to the cause of the dilatation.

On the other hand, special kinds of vomiting are pathognomonic.

1. *Vomiting of blood (hæmatemesis, melæna)* is pathognomonic of: *a. Ulcer of the stomach*, when the vomited matter is composed of fresh, dark-red, inodorous blood; it also occurs in cirrhosis of the liver. *b. Vomiting of old, decomposed, sometimes badly smelling blood (coffee-ground vomit)* is pathognomonic of carcinoma.

Blood is sometimes vomited when the gastric mucous membrane is inflamed by irritating substances, such as acids and alkalies. In hæmophilia blood may be vomited without an essential cause. Hysterical women in rare cases have hæmatemesis. In young girls vomiting of blood at the time of a suppression of the menstrual flow is of no serious consequence (vicarious hæmatemesis). Still one should always examine carefully for ulcer of the stomach.

One should be on the watch to distinguish between vomiting of blood and coughing of blood. In the majority of cases patients will describe characteristically either hæmatemesis or hæmoptysis. But sometimes coughing is attended by retching, and sometimes blood which has been coughed up is swallowed. In some cases hæmoptysis or hæmatemesis is the first sign of a pulmonary or of a gastric disorder which may have been concealed until the appearance of this sign startles the patient to the highest degree so that he cannot describe accurately the manner in which it appeared. In such cases (seldom occurring) it may be much more

difficult to establish a differential diagnosis than in cases of fresh bleeding, where it should be always a rule to *exercise the greatest care in conducting the examination of the organ, or to postpone it until all hæmorrhage has ceased for some time.*

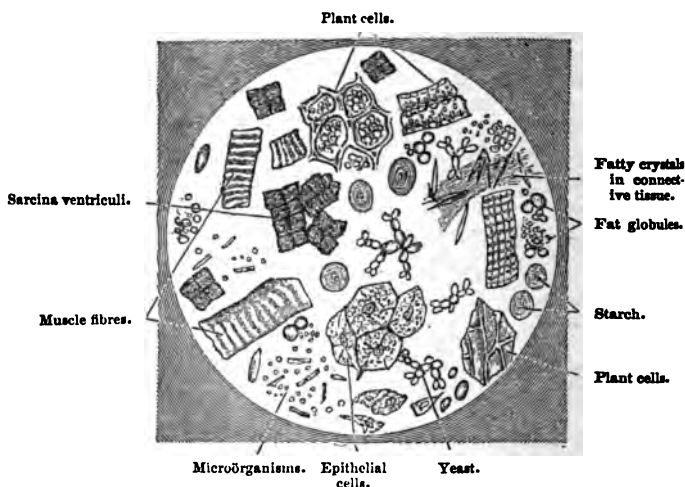


FIG. 23. — SCHEMATIC MICROSCOPIC PICTURE OF VOMITUS.

2. *Fæcal vomiting (miserere)* is the sign of intestinal obstruction (*ileus*) (see p. 95.)

3. *Very copious vomiting* appearing after long intervals of time is characteristic of *dilatation of the stomach*.

In a dilated stomach the food accumulates, owing to the atony of the walls or to the inability to pass into the pylorus on account of mechanical obstacles. As soon as the stomach is over-distended by reason of the continuous ingestion of food, it gets rid of a part of its contents by the vomiting of 1 or 2 litres of often decomposed fermented material. After this the patient feels better, eats for several days with steadily increasing discomfort until finally very copious vomiting begins anew.

4. *Matutinal vomiting* or morning vomiting before breakfast, accompanied by intense nausea and composed mostly

of mucus and only seldom of clear fluid, is produced by a pharyngitis such as is most frequently found combined with an alcoholic gastritis (*vomitus matutinus potatorum*).

5. Vomiting *immediately after eating*, chiefly attended by nausea, is characteristic of hysterical or nervous dyspepsia. In such cases one should look always for other signs of neurasthenia.

In most cases it is not possible to make a definite diagnosis from the character of the complaints of the dyspeptics. Under these circumstances it is necessary to obtain an

OBJECTIVE EXAMINATION OF THE STOMACH

The estimation of the general condition of the patient is of the greatest value. Rapid loss of strength would indicate a gastric carcinoma, whereas a good appearance would contra-indicate it; still extensive emaciation may accompany chronic gastric catarrh and dilatation of the stomach. One should notice the attitude of the patient, the character of his complaints, the expression of his face and of his eyes in order to discover whether he is of nervous temperament.

Inspection is generally of little value. Only in cases of considerable dilatation does the stomach appear as a distended bladder extruding the thin abdominal wall.

Palpation. — On palpation one should be on the watch for

1. *Tenderness*, which accompanies many of these disorders. Only severe localized pain which is increased on pressure would indicate ulcer.

The pain of ulcer of the stomach is especially characterized by the fact that it is produced by the contact of the ulcer with the acid contents of the stomach and increases after food has been taken. When the stomach is free from food in cases of ulcer, the patients are as a rule free from pain. The pain begins soon after eating, increases gradually, and reaches its highest point from 2 to 3 hours after the introduction of food. Vomiting the acid stomach contents produces a diminution of the pain. It is often possible to determine the *location of the ulcer* from the pain, which changes with different positions of the body; patients with an ulcer of the pylorus are relieved of pain by lying on the left side, an act which takes the food away from the pyloric end; should the pain be

situated on the posterior wall of the stomach, an abdominal posture will relieve it. *Ulcer of the duodenum* is recognized by the fact that the pain begins only after the food contents have passed into the small intestine in from 2 to 3 hours after ingestion.

2. *The presence of a tumor.* Only when a tumor can be felt ought we make a diagnosis of carcinoma. Tumors of the stomach move only slightly on respiration, whereas hepatic tumors move up and down distinctly. Should no tumor be felt when cancer is supposed to exist, then the diagnosis of carcinoma should be made tentatively; for one may be present on the posterior wall which can not be felt by palpation.

Tumors of the stomach are mostly carcinoma when they occur in old people. Still, there are practically many important exceptions, as: 1. *hypertrophy of the pylorus*, which may feel like a tumor about the size of a pigeon's egg. The diagnosis of this disorder is confirmed by the presence of the signs of ulcer, the long duration of the symptoms, the, as a rule, good preservation of the body, and the failure of the tumor to grow in size. Still, when accompanied by an extensive dilatation, the differential diagnosis may be most difficult. 2. *Perigastritis in chronic gastric ulcer.* Chronic ulcer of the stomach leads frequently to a diffuse infiltration and thickening of the tissues about the ulcer, which may give rise on palpation to the sensation of a tumor. Here, also, a fairly well nourished condition of the body, the long duration of the disease, the preceding symptoms of ulcer, and the structure (consistency) of the tumor may lead to the diagnosis.

Percussion. — By its means the size of the stomach is determined, though simple percussion, as a rule, gives untrustworthy results, because the surrounding intestines give rise to the same percussion note (Fig. 24).

The stomach is so situated in the abdominal cavity that $\frac{2}{3}$ of its volume lie on the left side of the median line, while $\frac{1}{3}$ is on the right of that line. The fundus lies in the concavity of the left vault of the diaphragm, the cardia at the level of the 11th to 12th dorsal vertebra; the small curvature and the pylorus are covered by the liver. The pylorus is situated on the right sternal line at the

level of the apex of the xiphoid process. The lower limits of the stomach extend to from 2 to 3 finger breadths above the umbilicus. The space, within which the tympanic note of the stomach is heard, over the thoracic wall, is called the *semilunar space*; the boundaries of the semilunar space are liver, lung, spleen, and arch of the ribs.

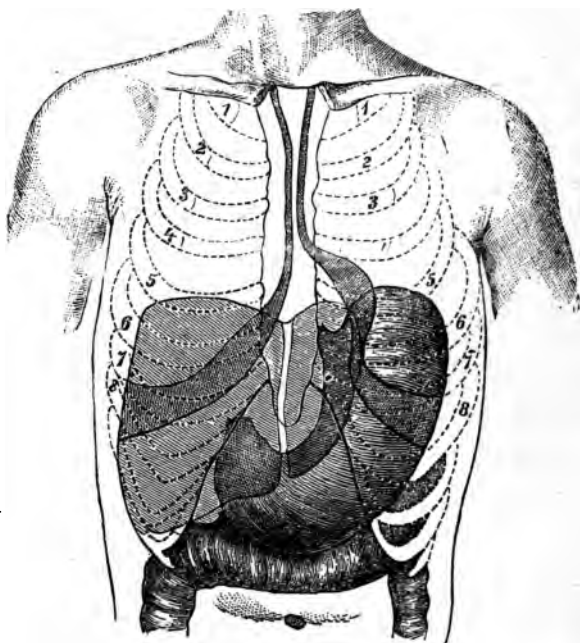


FIG. 24. — RELATIVE POSITION OF STOMACH, LIVER, AND COLON.

Valuable results are obtained by percussing the stomach when, after its being empty, several glasses of water are taken one after the other during definite intervals. In such a case, on percussing, a gradually increasing area of dulness is obtained corresponding to the drinking of each glass and by which the lower border of the stomach may be distinctly recognized.

The most reliable results in reference to determining the size of the stomach are to be obtained by

BALLOONING THE STOMACH

This ought to be done in all cases in which a suspicion of a dilatation of the stomach is entertained. Should there be a suspicion of the presence of an ulcer, distending the stomach with gas should not be performed, as it would be extremely hazardous.

The operation is performed by administering to the patient when the stomach is empty a teaspoonful of tartaric acid, and following this with the administration of a teaspoonful of sodium bicarbonate dissolved in a little water. Carbonic acid gas is thus generated within the stomach, and as a result the contours of the stomach in many cases become distinctly visible within the abdominal wall. In other cases where the stomach is not thus forced out so visibly, percussion of the organ easily differentiates it from the surrounding viscera. Should the disease be one which would indicate the introduction of a stomach-tube, the stomach may be directly blown up by the careful use of an air-pump.

The stomach is the seat of *dilatation* when its lower border reaches the umbilicus.

It should be observed, however, that there are many people who have naturally an unusually large stomach, which, nevertheless, performs its functions well (*megalograstia*). *Dilatation of the stomach* only refers to those cases of gastric enlargement which are produced by pathological causes and which give rise to disease symptoms. A descent of the lower border of the stomach may be produced by a dislocation of the entire organ; in these cases the transverse colon, as a rule, is also shoved downwards and the other abdominal viscera, such as the kidneys, are displaced, owing to the relaxation of the peritoneal folds (*enteroptosis*, *Glénard's* disease).

Recently the size of the stomach has been determined by the introduction into the organ of an electric light, which illuminates it so that its contours may be mapped out (*gastrodiaphany*). This method has also been used for diagnosing the presence of a tumor

of the stomach. Its use, however, necessitates an expensive instrument and a very strong electric current.

By the use of these methods of examination a differential diagnosis may be made in very many cases.

In several cases the determination of a diagnosis offers many data for the necessary therapeutics; *e.g.*, ulcer and dilatation. In a large class of cases, nevertheless, the treatment depends on the cause of the conditions of the acidity of the stomach. An anatomical diagnosis can only determine this in a few cases, because many cases of the same form of disease must be treated differently according as the acidity of the gastric juices varies.

The treatment of many diseases of the stomach, especially of catarrhs and neuroses, is chiefly a dietetic one. But the regulation of the diet is directly dependent upon the conditions of acidity. Patients with a hydrochloric hyperacidity bear meat extremely well, milk also in most cases, but digest the carbohydrates badly and only partially the fats. Patients with an anacidity digest fat and carbohydrates well as a rule if fermentation is checked, whereas meat in large quantities distresses them easily. The medicinal treatment—whether it should be an acid or an alkali—is directly dependent on the knowledge of the acidity of the stomach. Finally, the indications for washing out the stomach may only be first determined by an examination of the stomach contents, as, for instance, in organic hyperacidity.

In all those cases where the anatomical diagnosis presents no sufficient data for the selection of a mode of treatment, we should undertake

THE ANALYSIS OF THE CONTENTS OF THE STOMACH

Methods and normal relation. In the morning, when the stomach is entirely empty, a soft stomach-tube should be introduced, and by means of a small aspirating bottle some of the contents is aspirated. The healthy stomach before breakfast contains nothing or but a little slightly acid fluid. Accordingly the patient should be instructed to drink a pint of milk and to eat some bread (milk test meal). Two hours thereafter the stomach-tube is again intro-

duced, and the contents of the stomach are aspirated. These should be filtered, and the filtered solution examined as to its acidity. The introduction of the tube is easily done without much trouble to the patient; still, a certain skill is required in introducing it which may be acquired by practice. Should the retching which follows the attempt at introduction be excessive, a 10 per cent. solution of cocaine may be applied by a brush to the pharynx about 10 minutes before operating. Introducing a tube in an empty stomach is not necessary in all cases, but is a desirable procedure in cases of hyperacidity.

Instead of the milk test meal, the patient may take: 1. *Ewald's* test meal, composed of a roll and a cup of tea. This should be aspirated from the stomach for test purposes in three-quarters of an hour. 2. The *Leube-Riegel* test meal, which consists of barley-soup, 150 to 200 grammes of beefsteak, 50 grammes of bread and a glass of water, aspirated in 4 to 5 hours. 3. Should an accurate determination of whether *lactic acid* is formed in the stomach be required, it is best to use *Boas's* suggestion of giving, the night before the stomach is to be aspirated, a meal composed of oatmeal (1 tablespoonful of oatmeal, 1 teaspoonful of salt, and 1 quart of water, boiled together). The following morning an aspiration of the stomach should be made.

Instead of aspirating we may gain sufficient of the stomach contents by pressing upon the epigastrium, and having the patient at the same time attempt to vomit (*Ewald's* expression method).

The contents are filtered and the filtered solution is examined:—

1. With litmus paper.

2. For *free acid*. Pour a few drops of a watery solution of *methyl-violet* into a watch glass full of the stomach contents. If but a little hydrochloric acid be present, the solution becomes slightly blue. Another test is to add a few drops of a weak solution of yellow tropæolin; in the presence of free HCl the solution becomes more or less red. A third test is with *Günzburg's* solution, composed of *phloroglucin*, 2 grammes; *vanillin*, 1 gramme; *absolute alcohol*, 30 grammes (to be preserved in a dark bottle), of which one drop is added to several of the gastric fluid in a porcelain dish which is put over a Bunsen burner and slightly warmed; if there be any hydrochloric acid present, red streaks composed of the finest crystals develop.

3. For lactic acid with *Uffelmann's* solution, which is composed of 10 c.c. of a 1 per cent. solution of carbolic acid, 1 to 2 drops of iron perchlorate, which makes a blue-violet color. To this

reagent, which may be placed in a test tube, a few drops of the gastric fluid are added; if a large quantity of lactic acid be present, the fluid becomes yellow. In the presence of a large quantity of hydrochloric acid this test will fail, so that it would be necessary to shake 10 c.c. of gastric fluid and 50 c.c. of ether in order to obtain the lactic acid.

4. For *peptone* with the *biuret* reaction; a specimen of gastric fluid is rendered strongly alkaline with sodium hydrate, a 1 per cent. cupric sulphate solution is added drop by drop, and if *peptone* be present a distinct red color will appear.

5. It is necessary to determine the *total acidity*. For this purpose 10 c.c. of gastric fluid are titrated with deci-normal sodium hydrate solution. It is advisable to use a graduated burette, from which the deci-normal sodium hydrate solution may be dropped, and a pipette with which 10 c.c. of gastric fluid may be put into a glass beaker. The gastric fluid in the beaker is diluted with distilled water until it is nearly colorless, and 2 drops of an alcoholic solution of *phenolphthalein* are added. This solution becomes red in alkaline solutions, while it remains colorless in neutral and acid solutions. After this, one should carefully drop from the burette the sodium hydrate solution into the diluted gastric fluid until the mixture becomes of a faint rose tint. The amount of deci-normal sodium hydrate used indicates the amount of acid the stomach fluid contained; for example, to produce the rose tint in the 10 c.c. of gastric fluid 5.8 c.c. of sodium hydrate were used; therefore in 100 c.c. of gastric fluid 58 c.c. would have been used; the total acidity is hence 58. But since 1 c.c. of deci-normal sodium hydrate solution contains 0.004 g. of NaHO, the 58 acidity means that it would require 58×0.004 g. NaHO to neutralize the amount of acid present. Therefore $58 \times 0.004 = 0.232\%$ = total acidity, or 2.32 per thousand NaHO. Should this result be desired in terms of hydrochloric acid, we can utilize the formula $\text{NaHO} : \text{HCl} :: 40 : 36.5$, from which the acidity $58 = 0.212\% = 2.12\%$ HCl.

The determination of the total acidity is the most important feature of the examination. If the stomach was washed out thoroughly until clear fluid is returned, before the test meal, then the acid present after the test meal may be considered to be, for the greatest part, hydrochloric acid, even if the methyl-violet reaction is negative. The blue reaction of the methyl-violet is interfered with, as well ;

the other color reactions, should there be a large quantity of albuminoids in the fluid examined. The color reaction should be controlled by the biuret test; should this be positively present, then it is proven that hydrochloric acid is effective in changing albumin to peptone. From this result the conclusion is satisfactory that the *absence of the hydrochloric acid color reaction is no proof that there is no hydrochloric acid in the gastric fluid*. On the other hand, the presence of the color test indicates, with sufficient certainty that hydrochloric acid does exist in the fluid.

Should the stomach not have been washed out before the test meal, then the total acidity represents the combined hydrochloric and other organic acids (lactic, acetic, and butyric). By means of the methyl-violet, the biuret, and the Uffelmann tests, the valuable conclusion whether much organic acid is present is reached. Should it be—in especially important cases—desirable to determine quantitatively the amount of hydrochloric and lactic acids, complicated chemical methods must be employed.

In normal cases, the gastric fluid obtained two hours after a test meal is acid in reaction and responds to the litmus and to the hydrochloric acid tests, as well as to the biuret test, but gives no reaction to the test for lactic acid.

The total acidity varies between 50 and 65 (c.c. of normal sodium hydrate solution to 100 c.c. of filtered solution) 0.18 to 0.24 % of hydrochloric acid.

The hydrochloric acid reactions are absent in gastritis and in carcinoma of the stomach; they are often markedly increased in ulcer of the stomach and in nervous dyspepsia.

The failure of the hydrochloric acid reaction is by no means calculated to disprove the presence of carcinoma of the stomach; still, a marked presence of this reaction would contra-indicate, in most cases, the existence of that disease.

The proof of the presence of a large quantity of lac-

acid would strongly indicate carcinoma. The test, however, is only conclusive providing the stomach has been well washed out, and the test meal is deficient in food containing lactic acid (*Boas's* test meal, see p. 86).

In addition to the acids, the stomach contains the digestive ferments, *pepsin*, which peptonizes the albuminoids, *rennet ferment*, which coagulates milk, and their precursors, pepsinogen and labzymogen. The examination for these ferments has as yet no essential diagnostic significance, as they are present in most cases of stomach disease.

The consideration of the artificially obtained gastric fluid (the question of the relation between the amount thus obtained and the amount of food) justifies the conclusion as to how much food the stomach has passed into the intestine (the *motor activity* of the stomach). From this not infrequently a judgment as to the improvement or non-improvement of a case may be formed. According to *Leube* every stomach is deemed insufficient from which remnants of food may be obtained by aspiration seven hours after a meal.

A better method of determining the motor activity of the stomach is by means of the *salol test*. Salol passes from the stomach into the intestine unchanged, and is split up only by the alkaline secretion of the intestine into salicylic acid and phenol; the reaction of salicylic acid in the urine (a violet color on the addition of ferric chloride) indicates that salol has passed into the intestines. Two (2) grammes of salol should be given in a wafer at a meal. In the normal person, the reaction for salicylic acid will be obtained in the urine in $\frac{3}{4}$ to 1 hour afterwards, that is, urine thus obtained will show a violet color to ferric chloride. In motor weakness of the stomach this reaction will appear from 2 to 5 hours afterwards. In addition a distinct reaction may be obtained in cases of good motor function at the utmost up to 24 hours afterwards, while in motor insufficiency it will persist for 2 days.

It is desirable to determine more definitely in a few individually important cases, for instance in stricture of the pylorus, the motor activity of the stomach. For this purpose 100 c.c. of pure olive oil are poured into the stomach which has been previously washed out;

and after 2 hours an aspiration of the gastric fluid is made (*test*). The normal stomach will pass 50 to 75 c.c. of the 100 c.c. of oil that had been given into the intestine; in very many disease conditions the amount of oil obtained from the stomach is more or less diminished.

CHIEF SYMPTOMS OF THE MOST IMPORTANT DISEASES OF THE STOMACH

Acute gastritis.—Severe dyspeptic symptoms are prominent. It is caused chiefly by errors in diet. Vomiting is an accompaniment, together with diminution or absence of hydrochloric acid. The region of the stomach is tender. Not infrequently there is headache and some fever. The termination is mostly favorable, still a transition into a chronic gastric catarrh is possible.

Ulcer of the stomach (*ulcus ventriculi*).—Is manifested by *localized pain*, with vomiting after the ingestion of food, the vomited material being often strongly acid. Vomiting of blood also occurs (*hæmatemesis*). The condition of nutrition of the body is somewhat below par. The acidity is most always increased, still when anaemia is present it may be diminished.

Carcinoma of the stomach.—There is a palpable *tumor* of the organ. Vomiting of the contents of a coffee-ground appearance occurs; the patient is distinctly cachectic. The reaction to the test for hydrochloric acid is almost always absent, but lactic acid in the stomach contents is increased. The duration of the disease is between 2 and 3 years.

The dyspeptic symptoms which appear in old persons unaccompanied by a palpable tumor and by vomiting, and which in spite of a rational system of treatment persist and lead to excessive emaciation, may be mistaken for carcinoma of long standing.

Dilatation of the stomach.—At more or less long intervals vomiting of large quantities takes place. The lower border of the distended stomach lies below the umbilicus. The bowels are constipated, the amount of urine is small, and the skin is dry. Emaciation is noticeable.

The diagnosis should at the same time embrace the causative factor of the disease: whether the disorder is due to stricture of the pylorus or to atony of the muscular structure of the stomach. The cause of stricture is to be found in, either a cicatricial formation (due to an ulcer or to the inflammation resulting from caustic substances taken into the stomach) or in a carcinoma. The stric-

ure is sometimes produced in rare instances by the compression of a tumor, by twist or bend of the duodenum caused by peritoneal adhesions, by a floating kidney, etc. The atonic dilatation is the result of direct distention, due to excessive drinking and gluttony, and of chronic gastric catarrh.

Chronic gastritis.—Palpation and percussion of the stomach demonstrates, outside of the abnormal sensitiveness of the organ, no foundation for the severe dyspeptic symptoms. If proof of taking injurious substances into the organ can be deduced, the diagnosis of the disease is probable. The stomach contents contain much mucus, and the acidity is diminished (still in a few cases it is increased).

Nervous dyspepsia.—The symptoms which may be objectively demonstrated give no indication of the severity of the disorder. There is often diffuse pain over the stomach, mostly independent of the ingestion of food and the position of the body, and often hydrochloric hyperacidity. When a neurotic condition can be demonstrated and neurasthenic symptoms are present, the diagnosis is easy; it may also be made if the causes which lead to gastritis can be excluded.

DIAGNOSIS OF THE DISEASES OF THE INTESTINES AND THE PERITONEUM

The diseases of the intestines and of the peritoneum are recognized outside of the consideration of the general condition of the patient by an examination of the *fæces* and of the *abdomen*.

FÆCES

The normal person has one or two movements of the bowels in twenty-four hours. The stool is formed and of the consistency of thick mush. *Thin* or *fluid* stools constitute *diarrhæa*. (See p. 93.) The color and amount of the *fæces* depend upon the food. When meat has been the chief article of diet, the amount is small, the color is brownish, and the fecal mass is firm; when chiefly bread and potatoes are eaten, it is more voluminous, soft, and yellowish brown; when milk is exclusively taken, it is yellowish white, and tolerably firm.

The ordinary color of the *fæces* is caused partly by the reduced coloring matter of the bile (hydrobilirubin). The reduction is caused by the bacteria in the intestines.

Abnormally colored stools: *black stools* are the result of hæmorrhage (see below), or are occasioned by medicines such as iron, bismuth, which form respectively sulphide of iron and sulphide of bismuth. *Green stools* are produced by mercury, especially by *calomel*, which forms mercuric sulphide at the same time as it does biliverdin, or by passage through the intestine of unchanged bile, which occurs in the *summer diarrhœa* of children. *Grayish-white stools* occur in fæces free from bile, and when the fæces contain much fat.

Fatty stools are grayish-white, mucilaginous, and soapy; they occur as the result of the occlusion of bile from the intestine (icterus), and in addition chiefly from chronic peritonitis and intense anæmia.

Bloody stools appear as blackish-brown fæcal masses looking like tar, and are present in *enteritis*, tumors of the intestine, embolism of the mesenteric arteries, in typhoid fever and in purpura. Hæmorrhages from piles give rise to the bright-red color of the stool.

Purulent stools. Should the stool contain only pus, perforation of a peritoneal exudate into the intestine can be diagnosed; when only mixed in small quantities in the fæcal mass, and associated with fluid and at times bloody stools, it would indicate an *ulcer* of the large intestine. Bloody purulent stools, frequently in small amounts and accompanied by severe tenesmus, indicate *dysentery*.

Mucous stools. Pure mucus denotes *catarrh* of the large intestine, as does the encapsulation of the scybulous stool with mucus.

Small particles of mucus intimately mixed like grains (often like sago-grains, or only microscopically demonstrable) in the formed stool, indicate the presence of a *catarrh* of the small intestine. Sometimes these particles are colored with bile. Mucous formation in the shape of tubes (mucous casts) occurs in *mucous colic* (a secretory neurosis of the large intestine).

place. The series of causes just mentioned may also lead to deficient absorption. To this category belong especially (a) amyloid degeneration of the intestinal mucous membrane; (b) obstruction in the portal circulation (cirrhosis of the liver) and the general venous circulation (non-compensated cardiac disease). The obstructive conditions lead again to catarrh, and belong therefore in part to the first group.

3. By the transudation of watery fluids into the intestine; infectious and inflammatory states fall under this group.

Diarrhoea of short duration, mostly without significance, yet leading quickly under certain conditions to severe symptoms, occurs in cases of nervous or psychic excitement, exposure, in acute gastro-intestinal catarrh, and is infectious, especially when occurring in children. It occurs as a symptom of cholera, summer diarrhoea, and cholera nostras.

Diarrhoea of longer duration, chronic diarrhoea, occurs in portal obstruction; in interference with the general reticular circulation as in some forms of heart disease; in the various intoxications, e.g. uræmia, as well as in certain sub-acute infectious diseases, such as typhoid fever; in general toxic kind of diarrhoea justifies the diagnosis of a grave disorder of the intestine (either a chronic enteritis, a dysentery, a tubercular or syphilitic ulceration, or an amyloid degeneration). Tuberculosis of the intestine is only to be diagnosed with any degree of certainty when it can be proved to exist in other organs of the body, and amyloid degeneration only when one of its etiological factors, such as phthisis, syphilis, malaria, abscess, etc., is present. The situation of the ulcerative process may often be determined by the character of the mucus or pus mixed with the faecal discharges (see above). The following diseases have characteristic stools: *typhoid* fever, where the stool has a "peppery soup" appearance; *dysentery*, where it is mucous and bloody; and *cholera*, where it looks like "rice-water."

ABDOMEN

Normal relations. The abdomen of the healthy person is moderately arched, moves up and down during respiration, is usually soft, easily depressed, and nowhere painful on palpation; offers no resistance anywhere to pressure and is tympanitic on percussion excepting when the colon contains large faecal masses, when the percussion note may be dull.

Retraction of the abdomen. The abdomen is *scaphoid* in shape and retracted in spasm of the intestine such as occurs in colic and in meningitis, or when it is entirely empty as in inanition, in dilatation of the stomach, and in stricture of the œsophagus.

DISTENTION OF THE ABDOMEN

The abdomen may be highly arched, and sometimes tense as a drum. These conditions are caused by

I. An accumulation of gas in the intestines (**meteorism**, tympanites); determined by the general tympanitic resonance on percussion, the absence of fluctuation. Moderate tympanites occurs in typhoid fever, in chronic intestinal catarrh, and in obstruction in the portal circulation.

A *high degree* of tympanites occurs with (1) intestinal obstruction (*ileus*). The diagnosis of ileus is rendered certain when, in addition to extensive tympanites, there is the facies of collapse, small frequent pulse, and *faecal vomiting*. After the diagnosis of ileus is made, the following data must be determined: 1. The location of the obstruction; is it situated in the small or large intestine? 2. The nature of the obstruction, which may be an invagination, a twist (*volvulus*), a strangulated hernia, a constriction due to a peritoneal band, a peritoneal abscess, a cicatrix of an old intestinal ulceration, a malignant tumor, a foreign body such as a gall-stone.

The hernial canals and the rectum should always be examined without fail. Obstruction of the small intestine is accompanied by active visible peristaltic motion in the intestines, by a large amount of indican in the urine (Chap. VIII.), and by violent general symptoms; in obstruction in the large intestine, a great amount of distention of the portion of the colon above the constriction is observed, the urine contains but little indican, and the general symptoms develop more slowly. The diagnosis of the nature of the obstruction is made in many cases from the history of the case and by manual examination, but can not often be absolutely settled.

A high degree of tympanites occurs with (2) acute general peritonitis, where every movement of the patient is attended by pain, as is every touch on the abdomen; in addition there are present, vomiting of bile, a small frequent pulse, and the *facies hippocratica*.

Even in localized peritonitis an irritation of the entire peritoneum may result, so that the symptoms of a general peritonitis may be closely imitated. The diagnosis is determined by proof of the presence of an exudate.

II. By a collection of free fluid in the peritoneal cavity (*ascites*). In these cases the abdomen, when the patient is lying down, is distended at its sides and flattened in the middle; and there is tympanitic percussion, while dulness is elicited in dependent parts. Dulness on percussion is bounded above by a horizontal line and is changed by a change in the position of patient. On tapping the side of the abdomen with the finger *fluctuation* is often elicited.

If the patient is placed on his side, a tympanitic percussion note is obtained over the side which is uppermost; should the position be changed to the recumbent one, dulness on percussion will be found on the side which had just shown a tympanitic note.

Should ascites have been diagnosed, the following possibilities should be regarded:—

1. Ascites, together with general oedema, occurs in cardiac and renal diseases, but the ascites is only a secondary development to the original oedema, and is of itself not of essential importance.

Still, in consequence of a long-continued ascites, oedema of the legs may follow as a result of the pressure of the fluid on the veins and of the anæmia.

2. Ascites may be unaccompanied by general oedema, only by a secondary oedema of the legs. In this case there is present either:—

(a) *Obstruction to the portal circulation* by reason of hepatic disease or occlusion of the portal vein; under these conditions the ascitic fluid contains very little albumin, and varies in *specific gravity* from 1006 to 1015.

(b) *Chronic peritonitis*. In this case the ascitic fluid contains

tains a larger percentage of albumin, and its specific gravity is 1018.

The specific gravity is measured by a urinometer, the fluid to be examined being at the temperature of the room. When taken at the temperature of the body, it is lower than under the former conditions; it is 1° of the urinometer lower for every 3° C. elevation above the temperature of the room. From the specific gravity the amount of albumin may be proximately determined, in this manner, according to *Reuss's* formula, $A = \frac{2}{3}(S - 1000) - 2.8$, where A represents the amount of albumin in per cent., S , the specific gravity.

To return to (a), which we might call congestive ascites, it occurs chiefly in diseases of the liver, and especially in *hepatic cirrhosis*. Accompanying the ascites is a distention of the veins of the abdomen, especially those about the umbilicus (forming the *caput medusæ*); in addition the spleen is enlarged, and the stomach and intestines are the seat of a catarrhal inflammation. From the history will be elicited that the patient has indulged excessively in alcohol. In rare instances syphilis produces a peculiar variety of hepatic cirrhosis (*hepar lobatum*), which proceeds without ascites, and always runs a chronic course.

The other diseases of the liver, especially carcinoma and syphilis, give rise only seldom to ascites, and are diagnosed by the absence of the characteristic signs of cirrhosis, by palpation, and by the history.

Occlusion of the portal vein occurs very rarely, and is then produced by tumors of the stomach, of the pancreas, etc., or by a thrombosis of the vein.

(b) *Chronic peritonitis* is produced by carcinomatosis or by tuberculosis of the peritoneum, is accompanied by general cachexia, and the diagnosis is made positive when either carcinoma or tuberculosis is discovered in one or more of the other organs.

In chronic peritonitis the ascites may be encapsulated by peritoneal adhesions. In such a case the change of the position of the patient may not give rise to change in the percussion note. *Fric-*

tion sounds may be heard and friction be felt. Occasionally infiltration of the abdominal wall around the umbilicus is felt (*periomphalitis*).

III. *By tumors*, in which case the abdominal distension is often asymmetrical, being most prominent, not infrequently, at the site of the organ affected. Over the distended area dulness on percussion is present. Tympany may occur at the same time. A diagnosis of tumor can only be certain when a *tumor can be felt*.

We should be on our guard not to confound tumor with accumulation of feces in the intestine. In the latter case the mass is doughy in consistency, easily indented, and disappears after free catharsis.

Tumors may arise in the liver, the spleen, the kidneys, intestines, the stomach, the omentum, and only seldom in the vertebra and the pelvic bones, in the aorta (pulsating aneurysm) and in the female genital organs.

A tumor in the *ileocaecal region* of the size of an egg or orange, or even up to a saucer, if tender and combined with fever, vomiting, and tympanites, denotes the presence of *appendicitis* or perityphlitic exudation.

Tumors of the ovary and the pregnant uterus will show dullness on percussion over the lower half of the abdomen; the upper boundary of dulness is convex upwards, and in the lateral part of the abdomen in the recumbent position it is tympanitic; there is no change in percussion note.

To determine from which organ the tumor arises is often extremely difficult. To assist in the diagnosis, various tests are called into requisition, such as ballooning the stomach or filling it with water, or by doing the same to the cecum by means of a rectal tube.

IV. *By the escape of gas* into the peritoneal cavity. Gas within the abdominal cavity will always rise to the top, so that when the patient lies on the left side, the area of liver dullness will disappear, and when on the right side, no splenic dullness can be elicited. Gas within the peritoneal cavity

is the pathognomonic sign of a *perforation peritonitis*, which is almost invariably fatal.

Perforation of the stomach may be produced by a long-standing ulcer of the stomach, especially after an intense bodily strain, or after a full meal; perforation of the intestine may result from an ulcerative process in that viscus, especially occurring in the remission stage of typhoid fever, and after there has been a hæmorrhage from the bowel. Another frequent cause of perforation peritonitis is the perforation of the vermiform appendix by a fæcal concretion.

The *disappearance of liver dulness* has a fatal significance only when at the same time a change in position of the patient produces a change of percussion note; changing the patient's position, however, occasions intense pain, and should be done only in cases of urgent necessity. Perforation peritonitis is also made apparent by the signs of great collapse, as well as by the local signs.

The liver dulness is absent frequently in ordinary tympanites of moderate amount, such as occurs in constipation; in addition, it may be absent or much diminished in diseases of the liver, which lead to atrophy, such as acute yellow atrophy and cirrhosis. In acute atrophy of the liver a progressive diminution in liver dulness may be seen taking place from day to day. It is diminished in cases of extensive pulmonary emphysema, where the distended lungs overlap the liver area.

CHIEF SYMPTOMS OF THE MOST IMPORTANT DISEASES OF THE INTESTINES AND ABDOMEN

Acute intestinal catarrh: suddenly developing diarrhœa, with colic and tenesmus. In severe cases the final evacuations contain much watery mucus. It is often preceded by vomiting, and is sometimes accompanied by fever. There is almost always more or less depression. *Chronic intestinal catarrh* is characterized by a diarrhœa, which lasts for months, consisting of mucus, pus and bloody stools, alternating with constipation, slow emaciation. Specific causes, such as tuberculosis, syphilis, dysentery, and carcinoma, which may likewise be the cause of a chronic catarrh, should be excluded in making a diagnosis.

Mucous colic (formerly known as *enteritis membranacea*) is a rare form of intestinal affection, occurring in neuropathic individuals, and characterized by a diarrhoea accompanied by attacks of colic, the stools containing tape-like, cylindrical, pseudomembranous casts of coagulated mucus.

Carcinoma of the intestine: cachexia, and a palpable tumor in the abdomen, which is often very movable. The intestine may be moved with the tumor. It is frequently associated with signs of intestinal obstruction (see p. 95) and with hæmorrhages from the bowels. Tumors of the rectum can usually be felt from the anus.

Ileus. (See p. 95.)

Acute general peritonitis. There is extensive tympanites, vomiting of bile (spinach-green vomiting), at times singultus. The abdomen is intensely tender, and when due to a perforation the liver dulness gives place to tympanitic percussion, owing to the contained gas rising to that region. The facies of collapse are well marked, the pulse is frequent and small, and respiration is shallow and rapid.

Localized peritonitis. The general symptoms approximate more or less those of general peritonitis, though as a rule they are much less intense. By palpation a localized exudation can be felt. The rectum should be examined by the finger for this purpose.

Perityphlitis, appendicitis. A painful infiltration or exudation in the ileocæcal region may be felt. On percussion it is dull. There is vomiting, tympanites, constipation, and fever. The pulse is good, as a rule. It ends frequently in spontaneous absorption, though rupture of the abscess occurs; operation is not infrequently necessary.

Chronic peritonitis. (See p. 97.)

DIAGNOSIS OF DISEASES OF THE LIVER

The important data to be obtained in simple jaundice (icterus) are: mistakes in diet, previous gastric catarrh, fright, anger; when severer symptoms are present the following: a previous attack of

jaundice, especially of biliary colic (gall-stone); a history of alcoholism (cirrhosis); of companionship with dogs (echinococcus); of syphilis. One should determine also whether the etiological factors of amyloid degeneration are present and whether there has been any history of poisoning, for instance, with phosphorus.

Diseases of the liver are recognized in many cases by the presence of *icterus*, which is first observed in the conjunctiva and finally over the skin of the entire body. The urine is of the color of dark beer, shows *Gmelin's* reaction (see Chap. VIII.); the fæces are *grayish-white*, like clay. The following types are recognized:—

1. *Icterus simplex* (catarrhal jaundice), which is associated with mild symptoms, headache, fatigue, malaise, itching of the skin, and a slow pulse. It arises as the result of the occlusion of the ductus choledochus by a catarrhal inflammation of its mucous membrane. It lasts but a few weeks. The prognosis under proper treatment is favorable.

2. *Icterus gravis* occurs with severe symptoms of disease, with emaciation, frequently with fever, mental confusion, delirium, and severe pains in the region of the liver. It may be produced by passage of a gall-stone or impacted gall-stone, by liver abscess, by echinococcus of the liver, by carcinoma of that organ, and by acute yellow atrophy.

Concerning icterus with polycholia and without polycholia, see p. 8. The clinical consideration of cases of severe jaundice may present many prominent symptom-combinations which serve to make the diagnosis easy; thus icterus combined with *cachexia* indicates *hepatic carcinoma*; when combined with *ascites* it indicates *cirrhosis*; icterus occurring with *attacks of colic* indicates *biliary calculi*; when associated with *chilly sensations*, *abscess of the liver* is probably present. Of course these symptom-combinations have only a limited diagnostic value; they should always be corroborated by the other signs of the individual diseases elicited only by a careful examination. Thus, the picture of a jaundice due to gall-stone may be simulated by a carcinoma of the duodenum and that of cirrhosis of the liver by a chronic peritonitis, etc.

There are some diseases of the liver in which jaundice appears only late in the course of the disease or *not at all*. They are amyloid liver, fatty liver, congestive liver (due to impeded venous circulation), carcinoma of the liver, syphilitic liver, atrophic cirrhosis, and echinococcus. The attention of the clinician is drawn to the liver chiefly by the patient's symptoms of pressure and pain in the hepatic region, sometimes by the ascites, and often only after a casual physical examination of the organ.

A differential diagnosis is made from the history of the case and the general condition of the patient, and by the physical signs obtained by *percussion* and *palpation* of the organ.

The liver (compare Fig. 24, p. 83) lies in the right hypochondrium and its *upper border* in the normal state reaches the inferior border of the 7th rib in the axillary line; in the mamillary line it reaches the inferior border of the 6th or the superior border of the 7th rib; its upper border lies behind the 6th rib at the right sternal line. Its *inferior border* in the axillary line lies between the 10th and 11th ribs, *extends across the costal arch in the mamillary line*, and lies midway in the linea alba between the xiphoid process and the umbilicus; it then curves upwards and reaches the diaphragm at a spot between the parasternal and mamillary lines. On deep inspiration the liver descends a trifle.

Percussion of the liver will show relative dulness in the mamillary line from the 4th rib downwards, which changes to absolute dulness (flatness) at the lower border of the 6th rib. The latter ends in the mamillary line at the free border of the ribs, when the percussion note becomes tympanitic. *Palpation* gives no results in the normal condition of the organ in the mamillary line. But in *hypertrophy* of the liver the edge of the liver may be felt below the free border of the ribs and percussion will show dulness also below the free border.

Liver dulness is *increased* always in hypertrophic cirrhosis, in amyloid degeneration of the liver, in chronic congestion of the liver, often fatty degeneration of the liver, in hepatic echinococcus, carcinoma, and abscess.

Liver dulness may extend beyond the free border of the ribs even when the liver is not increased in size, providing the *diaphragm* is *shoved downwards*, as occurs in pulmonary emphysema, in pneumothorax, and in pleuritic effusions on the right side.

Liver dulness is *diminished* in area in acute yellow atrophy (here the diminution goes on from day to day, and without an increase of tympanites), in atrophic cirrhosis (diminution proceeds extremely slow), *often in tympanites* when the transverse colon lies between the liver and the abdominal parietes; should extensive tympanites coexist with grave general symptoms, and in addition should it be possible to elicit by a change of the position of the patient to the right side a liver dulness which was absent when the patient was on the left side, the diagnosis of air or gas in the peritoneal cavity should be made (perforation peritonitis, see pp. 98, 99).

The border of the liver is *felt* to be *smooth* in chronic congestion, in amyloid degeneration, and in hypertrophic cirrhosis. The border and the upper surface are *rough* (uneven and nodular) in atrophic cirrhosis, in syphilitic hepatic disease, in carcinoma, and sometimes in hepatic abscess.

A special form of liver tumor is the *corset or tight-lace liver* (constricted liver), where a portion of the right lobe lies below the free border of the ribs as an isolated tumor extending from 4 to 6 cm. into the abdominal cavity. Abnormal mobility of the liver is known as *floating liver* (it occurs in women with pendulous abdomens).

CHIEF SYMPTOMS OF THE MOST IMPORTANT DISEASES OF THE LIVER

Catarrh of the bile-ducts (catarrhal jaundice) is characterized by icterus, accompanied by mild symptoms (see pp. 8 and 101); the liver is often enlarged, is but little tender, and the gall-bladder is often palpable. A favorable termination occurs in from 3 to 5 weeks.

In a few cases it progresses to *chronic* jaundice by reason of the **agglutination** of the mucous walls of the ductus choledochus and

the occlusion of its canal (*cholangitis chronica fibrosa*). As a result gradual slow emaciation occurs and after a few years, death, preceded by coma and convulsions, ensues.

Abscess of the liver gives the symptoms of jaundice, combined with erratic chills, emaciation, severe pains in the region of the liver and in the right shoulder. In solitary abscess a protrusion of the liver surface upwards or downwards is sometimes observed. In multiple abscesses the liver is not infrequently enlarged throughout.

Biliary colic (gall-stone colic) is accompanied by very severe attacks of pain in the hepatic region of varying duration and occasionally by jaundice; vomiting and fever are not infrequent. The diagnosis is rendered positive by the finding of gall-stones in the fæces.

Analysis of gall-stones. Gall-stones are composed of concretions either of *bilirubin* or of *cholesterin*. Cholesterin is tested for as follows: a portion of the calculus is dissolved in hot alcohol and the solution is then filtered; from the filtered solution, when cool, cholesterin forms in rhombic crystals. To continue the test, the crystals are dissolved in chloroform and concentrated sulphuric acid is added to the solution, when a beautiful deep-red color appears, which gradually changes to blue and finally green. Bilirubin is isolated by extracting from the remaining filtrate with warm chloroform, the filtrate having previously been rendered slightly acid with hydrochloric acid, when it is observed in the chloroform solution on the addition of fuming nitric acid (*Gmelin's test*).

Carcinoma of the liver give rise to cachexia, combined with icterus; palpable, nodular tumor in the liver region; usually decided enlargement of the liver without any enlargement of the spleen.

Acute yellow atrophy of the liver is diagnosed when a suddenly developing jaundice occurs associated with severe cerebral symptoms such as confusion, delirium, and coma; with a rapid diminution of liver dulness. Tyrosin and *leucin* are found in the urine, the excretion of urea is very

much diminished, the excretion of ammonia very much increased. It pursues a rapidly lethal course.

Acute yellow atrophy may appear after an initial stage of catarrhal jaundice lasting from 8 to 14 days.

Hypertrophic cirrhosis of the liver is characterized by the presence of jaundice, combined with a decidedly uniform enlargement of that viscus; alcohol and syphilis are the etiological factors. It is associated with a visible enlargement of the veins of the abdominal walls, with a hypertrophy of the spleen, and with gastro-intestinal catarrh; ordinarily it is unaccompanied by ascites.

Atrophic cirrhosis of the liver is diagnosed by the presence of ascites, the ascitic fluid having a low specific gravity; by the distention of the abdominal veins and an appreciable splenic enlargement. There is a slowly developing cachexia and sometimes jaundice. Symptoms of gastro-intestinal catarrh. The history will show excessive drinking of alcoholic beverages, more seldom syphilis or malaria or chronic peritonitis or cardiac disease.

Echinococcus of the liver is only to be diagnosed when by the growth of the cyst the liver becomes enlarged. In well-developed cases fluctuation and hydatid fremitus over the tense elastic tumor may be elicited. Aspiration of the tumor mass yields a fluid in which portions of the echinococcus membranous wall and the hooklets of the parasite may be found on microscopical examination. The fluid does not become turbid, or at best but very little, on heating (see Chap. XII.)

Amyloid degeneration of the liver.—There is a uniform firm enlargement of the organ and a cachexia. Proof of the etiological factors, such as phthisis, syphilis, suppuration, etc., will assist in the diagnosis. The spleen is enlarged and albuminuria and diarrhœa are present.

Chronic congestion of the liver also presents a uniformly enlarged liver. Dyspnœa and cyanosis are features and

point to the primary cause of the disease; namely, to cardiac or pulmonary affections.

THE SPLEEN

Enlargement of the spleen is an extremely important sign, upon which depends the diagnosis of many diseases.

The spleen is situated in the left hypochondrium. Under normal conditions *splenic dulness* extends from the 9th to the 11th rib, and from the linea costo-articularis (a line drawn from the left sterno-clavicular articulation to the apex of the 11th rib) to the vertebral column. Should the spleen enlarge, its area of dulness will be increased until it finally extends beyond the left costal arch. When the organ is decidedly enlarged, its edge *may be felt* as a sharp border, especially on deep inspiration. Palpation of the viscus is often painful.

A positive proof of enlargement of the spleen is only possible when the organ can be *felt* by *palpation*; it is often possible to demonstrate an enlargement by percussion, though the results of percussion are frequently deceptive on account of the variable amount of fæces in the colon.

The determination of **splenic enlargement** is indispensable in making a diagnosis of *typhoid fever*, of *malarial intermittent*, and of *splenic leucæmia*; it is desirable to demonstrate it to reach a diagnosis of *amyloid degeneration*, of *cirrhosis of the liver*, and of *hæmorrhagic infarction of the spleen*.

It may be enlarged in all infectious diseases; in addition to typhoid, in pyæmia, pneumonia, etc. Should it be so found with pneumonia, it will remain demonstrable until resolution is completed.

The spleen may be displaced downwards in left-sided pleural effusions, in pneumothorax, and in pulmonary emphysema. It is displaced upwards in tympanites, in ascites, and by tumors of the abdominal cavity. Percussion dulness disappears in perforation peritonitis when the patient lies on the right side, and in general is absent over the splenic region in cases of *floating spleen*.

CHAPTER V

DIAGNOSIS OF THE DISEASES OF THE UPPER AIR-PASSAGES (NOSE, THROAT, LARYNX)

The diseases of the nose, to which attention is directed by the discharge, pain, and, above all, the *occlusion* of the nose, may be recognized by inspection with the nasal speculum and palpation with the nasal probe. The symptomatology of all the diseases of the nose would overstep the bounds of this work, and only those symptoms will be considered which are of importance in internal medicine.

Headache, especially frontal, is a symptom of many diseases of the nose. It may be present in a mild degree in an ordinary cold, usually to be referred, however, to disease of the frontal cavity. Purulent processes in this cavity (empyema of the frontal sinus) may give rise to meningitis; next to inflammations of the middle ear, the nose is probably the most frequent source of meningeal infection.

Neuralgia of the branches of the trigeminus is not rarely seen in disease of the nasal accessory chambers, and has been observed especially in connection with disease of the antrum of Highmore.

Epistaxis demands an examination of the nose; but the constitutional causes must not be lost sight of (chlorosis, leucæmia, anæmia, cirrhosis of the liver, contracted kidney, etc.).

A foul smell in the nose is the sign of *ozæna*. The nasal cavity is broadened (atrophy of the turbinated bones), the mucous membrane thin, pale, covered with numerous crusts.

The peculiar bad odor clings to the dried secretion. The secretion may be expelled through the nasopharynx; therefore *ozæna* is always to be looked for when there is a putrid expectoration (bronchitis, gangrene of the lung).

Mouth-breathing, which appears in every case of occlusion of the nose and nasopharynx (by hypertrophic catarrh, polyps, deviations of the septum, post-nasal tumors, etc.), is often accompanied by disturbing sequelæ: snoring, restless sleep (nightmare), inflammatory processes of the upper air-passages, disturbances of digestion; in children, after a considerable duration, typical facies (open mouth, dull expression), deformities of the teeth, the jaws, even of the thorax.

Adenoid vegetations, hypertrophy of the lymphoid elements in the nasopharynx (faucial or 3d tonsils), exceedingly common in children, give the most exquisite examples of the disturbances of mouth-breathing. They are a frequent source of deafness and may retard the complete bodily and mental development of a child (*aprosopia nasalis*). May be diagnosticated by the child's facial expression; positive diagnosis by the palpation of nasopharynx with the index finger.

The symptoms usually disappear rapidly after the removal of the vegetations.

Nasal reflex neuroses. — These are reflexes called forth by diseases of the nose. Attacks of migraine and spasms of coughing, epileptic seizures, etc., occasionally disappear after the cure of nasal affections. The best known of these is *nasal asthma*: attacks of bronchial asthma dependent upon an irritation from a diseased nose. An existing asthma is only to be referred to the nose when irritation of a particular spot induces an attack while its anæsthesia (by cocaine) stops the attack. Surgical measures for the relief of the asthma may then be undertaken. The same safeguard of diagnosis should be applied to the other neuroses of the diseased nose.

Throat and tonsils.—The inspection of the throat with depressed tongue shows whether infectious processes are localized there (angina or diphtheria, Chap. II.). At the same time, the presence of a *chronic pharyngitis* should be noted (mucous membrane puffy, reddened, covered with secretion or atrophic, dry, occasionally covered with fine granulations); it is often caused by the same elements that help to produce a chronic gastritis (alcoholism, too much smoking, the eating of very hot or poorly masticated food); it is seen in those working in dust of any kind, in speakers and singers.

Insensibility of the fauces is frequently a sign of hysteria or of advanced alcoholism, and may assist in the diagnosis of a contemporaneous gastric affection as neurotic or alcoholic. *Hyperæsthesia*, excessive gagging movements on touching, in chronic pharyngitis, also frequently in drinkers.

Retropharyngeal abscess is recognized by a fluctuating, bulging mass in the posterior pharyngeal wall; there are severe general disturbance and high fever. Deglutition and respiration may be interfered with. The threatening symptoms disappear upon the emptying of the abscess.

The source of cryptogenetic sepsis should be sought in retropharyngeal abscess and purulent processes in the accessory sinuses of the nose.

GENERAL SYMPTOMATOLOGY OF THE DISEASES OF THE LARYNX

The symptoms of laryngeal diseases are:—

1. *Changes in the voice.*—In the great majority of the diseases of the larynx, the voice is changed. It becomes hoarse, rough, rasping, indistinct (dysphonia). Aphonia (absence of voice, speaking in whispers) is a sign of severe disease of the larynx (ulceration of the vocal cords), or of imperfect closure of the glottis (paralysis of the vocal cords). *Falsetto voice* is usually a purely functional disturbance, a defective formation of the voice, which may be overcome by methodical exercise in speaking.

Ventricular voice. A peculiar rough and rattling voice is produced by the employment of the false vocal cords (ventricular bands) for the true ones, frequently in consequence of the disturbance of the latter.

The *nasal voice* may be *open* or *closed*. The former is due to the absence of the closure of the nasopharyngeal opening in consequence of the paralysis of the soft palate (principally after *diphtheria*) or in consequence of ulcerative processes of the soft palate (generally by syphilis). Test-words for this variety are, for example, pump, mumps. When this type of nasal twang exists, fluids taken into the mouth will return in part through the nose. A *closed* nasal voice arises from total occlusion of the nose (chronic catarrh, polyps, etc.).

Diphthongia (diplophonia) is the simultaneous production of two notes of different pitch in speaking; it sometimes occurs in unilateral paralysis of the vocal cords, as well as in the presence of small tumors on the edge of a vocal cord which, in speaking, are pressed between the vocal cords.

A *threefold splitting of the voice* is a rare phenomenon, produced by pediculated polypi which lie beneath the glottis. In speaking they are first pressed between the vocal cords in the expiratory act and then above the glottis. A prolonged vowel is first pronounced clearly, then hoarsely and diphthongic, and finally clearly again.

2. *Disturbances of breathing.* — The laryngeal disease may lead to dyspnoea through narrowing of the glottis. Laryngeal dyspnoea is *inspiratory*, accompanied by *stridor* (a creaking sound the result of the labored, long-drawn breath). The number of respirations is diminished; all the accessory muscles of inspiration are employed (see p. 125); the sides of the neck, intercostal spaces, epigastrium, are drawn in. Expiration is short, inaudible. Laryngeal stenosis occurs in children most commonly in consequence of diphtheria (croup), but occasionally through simple acute laryngitis (pseudo-croup), because of the narrowness of the child's glottis; in adults it is always a sign of severe laryngeal affec-

tion (acute œdema of the glottis, diphtheria, bilateral posticus paralysis, etc.).

Inspiratory dyspnoea and stridor are the same in *tracheal* as in laryngeal stenosis. The differential points are :—

In laryngeal stenosis the larynx makes effectual respiratory movements (it goes upward in inspiration, downward in expiration); in tracheal stenosis it remains quiet or almost so. The voice is usually free in tracheal stenosis; in laryngeal stenosis it is very hoarse or there is aphonia. In laryngeal stenosis the head is usually thrown back; in tracheal stenosis the chin approaches the chest. Concerning thrills, see below.

3. **Pain** in the larynx is frequently complained of and is described as tickling, pressure, burning, or soreness; inflammatory processes, particularly, produce severe pains, which radiate toward the ear. Pain does not furnish any especial diagnostic points.

4. **Cough**.—A *laryngeal cough* is sometimes of a particularly loud and barking character, and can frequently not be distinguished from a cough emanating from the lungs. It is called the *croup cough*, although by no means characteristic of croup, since pseudo-croup and other affections are responsible for coughs of similar sound. Cough is most easily produced from the posterior wall of the larynx (intra-arytenoid region). Irritation of the ventricular bands and of the vocal cords does not evoke a cough so easily.

5. **Expectoration** is common to most diseases of the larynx, but does not furnish any characteristic data as to its origin in the larynx.

6. **Dysphagia** is always a sign of severe and advanced disease of the larynx, and is especially characteristic of the involvement of the epiglottis and the posterior wall. The laryngeal dyspnoea and the change in the voice prevent diagnostic confusion with difficulties in swallowing caused by œsophageal stenosis. The changes in the throat which the dysphagia produces are visible.

THE EXTERNAL EXAMINATION OF THE LARYNX (INSPECTION AND PALPATION)

This is not of great importance. The visible respiratory movements of the larynx and their diagnostic significance in stenosis have been discussed above.

When stridor is present, one can feel a *thrill* in that part of the neck occupied by the trachea, which can be appreciated alone or stronger on expiration, when the obstruction to respiration lies deep in the trachea. In laryngeal stenosis, on the other hand, the thrill is palpable exclusively, or in the vast majority of instances, on inspiration.

Visible or even palpable pulsation of the trachea, or even of the entire larynx, has been described as a sign of aneurysm of the aorta.

The vibrations of the vocal cords in speaking may be felt equally on both sides by placing the index fingers at the sides of the thyreoid cartilages. Weakness of the vibrations of one side speaks for disease of that side; in combination with other symptoms (*e.g.*, a threefold division of the voice, see above), this sign may be the guide for a far-reaching diagnosis without the aid of the laryngoscope; the diagnosis of disease of the larynx is usually accomplished, however, by

LARYNGOSCOPIC EXAMINATION

The sunlight or the light of a lamp is thrown upon the laryngeal mirror, placed obliquely against the uvula, by a head mirror or reflector. The laryngeal mirror then gives the picture of the larynx.

The normal picture in the laryngeal mirror shows above (anteriorly) the epiglottis, below the posterior wall of the larynx, the intra-arytenoid region, the two arytenoid cartilages, and above these, as a slight prominence, the cartilages of Santorini and Wrisberg (cunicula laryngis and cuneiform cartilage, respectively). At the sides, the picture is bounded by folds of mucous membrane, which extend from the epiglottis (above) to the arytenoid cartilages (ary-epiglottic ligaments); the middle of the picture is taken up by the true vocal cords, which pass antero-posteriorly (from above downwards) and are divided into two parts, the ligamentous, the anterior two-thirds (formed by the arytenoid cartilages), and

the cartilaginous, occupying the posterior third. The cleft between the cords is known as the rima glottidis, the anterior portion of which is called the vocal glottis, the posterior the respiratory glottis. The false vocal cords (ventricular bands) run parallel to and above the true cords, and are seen at the side in the picture. Between the two, lies the ventricle of Morgagni. The right vocal cord appears on the right side in the mirror, the left on the left side; there is no transposition; but the right vocal cord is, of course, to the left of the observer.

The laryngoscopic examination is not intended to estimate merely the normal appearance of the larynx, but above all the mobility of the vocal cords also; the patient must alternately phonate (say *ah*) and take deep inspirations. In phonation the epiglottis is raised and the view into the larynx facilitated. During inspiration the glottis is opened, the vocal cords are abducted; in phonation they are adducted, the glottis is closed.

The direct inspection of the larynx without a laryngoscope (*Kirstein's autoscopy*) is rendered possible by the use of the autoscope when the uppermost air-passages are rendered straight by a peculiar position of the head, when the root of the tongue is pressed forward, and when there is a simultaneous elevation of the glottis.

NORMAL FUNCTIONS OF THE MUSCLES AND NERVES OF THE LARYNX

The opening of the glottis is accomplished by the crico-arytenoid muscle, innervated by the recurrent laryngeal nerve; it acts as an abductor by drawing the vocal process of the arytenoid cartilage outward.

Closing of the glottis. The intra-arytenoid muscle (*arytænoideus transversus* and *obliquus*) closes the glottis by drawing the arytenoid cartilages together; the other closers of the glottis are the crico-arytenoid lateralis, the principal adductor, which turns the vocal process inward, and thyreo-arytenoid internus, whose course is in the vocal cords. These muscles are innervated by the recurrent nerve, except the *arytænoideus transversus*, which receives a few fibres from the superior laryngeal nerve.

Tension and elongation of the vocal cords are accomplished by the crico-thyroid muscle, the nerve supply of which comes from the external branch of the superior laryngeal nerve and which draws the thyreoid cartilage forward and downward toward the previously fixed cricoid cartilage.

Tension and shortening of the vocal cords: the thyreo-arytenoid internus muscle, supplied by the recurrent nerve.

There are two laryngeal nerves, therefore, the superior laryngeal, the inferior laryngeal, or recurrent, which are branches of the accessory nerve, and which contain motor and sensory fibres.

The *superior laryngeal nerve* supplies with its small external branch the crico-thyroid muscle; the large inner branch perforates the hyo-thyroid membrane and supplies the mucous membrane with sensory fibres. Its motor fibres are few in number, and go to the *arytænoideus transversus* and the muscles of the epiglottis (the *thyreo-epiglotticus*, which raises the epiglottis, and the *ary-epiglotticus*, which lowers it).

The *inferior laryngeal nerve* passes backward in the thoracic cavity, on the right side encircling the subclavian artery, on the left the arch of the aorta, passes to the larynx between oesophagus and trachea, and innervates with its internal branch the *crico-arytænoideus posticus* and the *arytænoideus transversus* muscles, and with its external branch all the remaining muscles of the larynx.

SYMPTOMS OF THE MOST IMPORTANT DISEASES OF THE LARYNX

Acute laryngitis.— Usually no fever (in children, irregular remittent fever). Hoarseness and pain in the throat; slight difficulty in swallowing. Mucous or muco-purulent expectoration, not characteristic. *Laryngoscopic examination:* redness and swelling of the mucous membrane, diffuse or circumscribed. Vocal cords reddened, occasionally superficial erosions. Vocal cords apparently small in consequence of the swelling of the ventricular bands.

Chronic laryngitis.— Pressure, scratching, etc., in the larynx, especially in singing, smoking, etc.; speech thick, hoarse; aphonia after continued speaking. Scanty, muco-purulent sputum. *Laryngoscopic examination:* dirty gray-red discoloration of the vocal cords, profuse secretion, occasional swelling of the follicles of the mucous membrane to the size of a grain of sand (*laryngitis granulosa* or *follicularis*). Very often, callous thickening, grayish-red in color, of the posterior wall of the larynx.

Tuberculosis of the larynx. — Usually secondary to pulmonary phthisis. Hoarseness, aphonia, pain, dysphagia. *Laryngoscopic examination*, — 1st stage: *tubercular infiltration*. Mucous membrane, puffy, swollen, pale, œdematous. 2d stage: *tubercular ulceration*. Irregular, confluent swellings with jagged, raised edges, passing into the depths. The pit of the ulcer covered with purulent secretion in which tubercle bacilli are present. Sites of predilection for tubercular changes are, first, the arytenoid cartilages and the intra-arytenoid space, next, the true and false vocal cords.

Syphilis of the larynx. — The complaints and subjective symptoms are usually not characteristic. *Laryngoscopically* one may differentiate. 1. *Early forms* (secondary phenomena): erythema of the larynx, sharply limited red spots; syphilitic laryngitis, usually not to be distinguished from non-specific chronic inflammation (history and examination for other syphilitic lesions, especially in the throat); *papules*, grayish-white, flat elevations on the inflamed mucous membrane (mucous patches); superficial ulcerations (sites of predilection: epiglottis, especially its border and lingual surface, the vocal cords). 2. *Late forms* (tertiary phenomena): circumscribed gummata and diffuse gummatous infiltrations characterized by rapid necrosis; the ulcers following the necrosis are deep and are sharply limited, but can not always be easily distinguished from tubercular ulcers. The differential diagnosis is established by the demonstration of an existing pulmonary phthisis (tubercle bacilli in the sputum), or of a previous syphilitic infection; in doubtful cases by the result of *antisymphilitic treatment*. Luetic ulcers may become very important by the strong contraction of radiating scars, which may be the source of very severe laryngeal stenosis.

Tumors of the larynx. — Usually involving the true or false vocal cords, pediculated (polyps) or sessile. Symptoms dependent upon the size and situation of the growth; there

usually hoarseness, often the sensation of a foreign body, pain, dyspnoea, sometimes dysphagia. It is essential to differentiate between benign and malignant growths (carcinoma, rarely sarcoma). The benign tumors, frequently with a smooth surface, are always circumscribed; carcinoma, on the contrary, usually not sharply limited, of irregular form, soon ulcerating; swelling of the lymph-glands demonstrable. The motility of the vocal cords soon diminished in the presence of a malignant growth. Age and condition of patient's strength important. Diagnosis often difficult, especially in the early stages, in which it is important on account of the therapeutic measures. Usually a positive diagnosis can be made only by the microscopic examination of a small piece of the tumor secured by excision. The special anatomical diagnosis of *benign* tumors (fibroma, papilloma, myoma, adenoma, etc.) is only to be made by the microscopic examination after extirpation or the removal of a small piece by excision.

Pachydermia laryngis are wart-like thickenings on the most posterior parts of the vocal cords, usually containing a depression on the surface, occasionally with gaps or clefts, and then not unlike carcinoma; usually accompanies chronic catarrh.

Spasm of the glottis (laryngo-spasm): spasmodic closure of the glottis leading to the point of suffocation and unconsciousness. In children, stands in etiological relation to rachitis; in adults, usually caused by hysteria, but may be due to organic disease of the central nervous system (laryngeal crises of locomotor ataxia).

PARALYSIS OF THE VOCAL CORDS

When of peripheral origin, paralysis of the vocal cords may be produced by pressure upon the recurrent nerve (by goitre, aneurysm of the aorta, carcinoma of the oesophagus).

d lymph-glands, tumors mediastinum, pericardium in consequence of a neurolethic or rheumatic); *al* origin: hysteria (*functional* paralysis of the larynx), result of *organic* disease nucleus of the accessory in the floor of the 4th le (in locomotor ataxia, e sclerosis, bulbar palsy, syringomyelia, cerebral s, etc.).

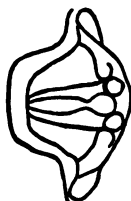
rentiation: 1. **Paralysis of the abductors of the vocal** - Paralysis of the extensors (crico-thyroid) recognized by palpation space between the thyroid and cricoid cartilages; the approach of the two in ion is absent. The glottis tense; it appears as a line; voice hoarse.

is a paralysis of the r laryngeal nerve (most tly a post-diphtheritic is), in which the depression of the epiglottis do not act tis immovable, usually d toward the root of the , and the sensibility of cious membrane of the is lost. These three (elevation of the epi-

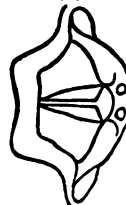
Bilateral paralysis of the recurrens.



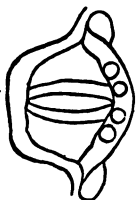
Paralysis of thyreo-arytenoid and intra-arytenoids.



Paralysis of the intra-arytenoids.



Paralysis of the thyreo-arytenoids.



Bilateral paralysis of the posticus.

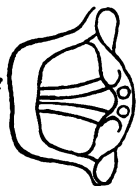


FIG. 25.

glottis, anæsthesia of the mucous membrane, atony of the glottis) are responsible for the dangers of mistakes in swallowing (foreign-body pneumonia).

2. **Paralysis of the adductors of the glottis.** — The paralysis of the adductors is usually functional. The glottis stands widely open (inspiratory position through action of the antagonistic muscles), when all three adductors are attacked (*crico-arytænoideus lateralis*, *thyreo-arytænoideus internus*, and *arytænoideus transversus* — complete paralysis of the adductors). The glottis then has the form of a triangle, in consequence of which there is no interference with respiration, but complete aphonia exists.

Isolated paralysis of the arytenoideus transversus muscle (frequent in acute catarrh and hysteria) shows the cartilaginous portion of the glottis gaping as a small triangle while the anterior part of the glottis closes in phonation. Hoarseness, even aphonia, without dyspnoea, supervenes.

Exclusive paralysis of the crico-arytænoideus lateralis is very rare and scarcely to be diagnosticated positively. On phonation a small cleft of the glottis near the vocal processes remains open; the disturbance of the voice is meagre.

Paralysis of the *thyreo-arytænoidei interni* is the most frequent; on phonation a small oval of the glottis, especially in its centre, remains open; the edges of the poorly vibrating, narrow vocal cords appear excavated; the voice is aphonic; no dyspnoea. This type of paralysis is common as an *occupational neurosis* (singers, public speakers, etc.), and usually presents the picture of *hysterical aphonia* (rarely total paralysis of the adductors).

Functional hysterical aphonia may be recognized by its sudden appearance and disappearance (intermittent aphonia is almost always hysterical). It may accompany psychical disorders; the cough usually has a ringing tone. Mere pressure upon the thyreoid cartilages produces the peculiar tone of the cough. The history and the other symptoms render the diagnosis that of hysteria.

3. **Paralysis of the abductors of the glottis** (*posticus paralysis*). — The paralysis of these muscles is always organic, never functional. In paralysis of the *crico-arytæ-noideus posticus* muscle, the vocal cord lies in the median line, through the action of antagonistic muscles; phonation is therefore more or less intact.

Unilateral abductor paralysis: no dyspnœa; voice sometimes rattling and not clear, more frequently intact; no symptoms then arise, but the paralysis is accidentally discovered (in syringomyelia, tabes dorsalis, etc.).

Bilateral abductor paralysis: both vocal cords immovable, lying in or very near the median line (stenosis of the glottis); voice almost normal, but severe inspiratory dyspnœa with stridor. Expiration easy. The closely lying vocal cords approach each other more closely in inspiration. Tracheotomy necessary.

4. **Paralysis of the recurrent laryngeal nerve.** — If all the branches of the recurrent nerve are paralyzed, adduction and abduction stopped, the vocal cords take a position between the two: *cadaveric position*.

Unilateral paralysis of the recurrent nerve. The paralyzed vocal cord in cadaveric position with excavated edge; the arytenoid cartilage dropped forward toward the interior of the larynx; the unaffected cord often passes beyond the median line in phonation, so that a closure of the oblique glottis supervenes (over-riding of the arytenoid cartilages); voice weak, with no ring; no dyspnœa.

Bilateral paralysis of the recurrent nerve. Both vocal cords in cadaveric position, glottis widely opened in the same position in phonation and respiration. *Absolute aphonia* (cough and expectoration weak, and with little sound); no dyspnœa.

In the recurrent nerve, the adductor and abductor filaments lie separated. The abducting nerves are physiologically more sensitive than the adductors. All influences

which lead to recurrent paralysis (gôitres, aneurysms, etc., see above) frequently cause a primary abductor paralysis which after a longer or shorter time (sometimes years) passes into a recurrent paralysis as soon as the adductor part of the nerve suffers from the disease.

CHAPTER VI

THE DIAGNOSIS OF DISEASES OF THE RESPIRATORY TRACT

IN the history of the patient, particular stress must be laid upon *hereditary taint* when tuberculosis is suspected. The so-called *scrofulous* affections of childhood and previous pulmonary diseases are important, as are *hæmatemeses* and inflammations of the bones or joints of a probable tubercular origin. The occupation (stone-cutters, coal-miners, compositors, etc.) must also be taken into account, and sometimes a weakening condition, such as a recent puerperium, may offer a basis for pulmonary invasion. When lung trouble of gradual origin is before the physician, the early symptoms of phthisis pulmonalis must be looked for.

The attention of the physician is directed toward the respiratory apparatus on complaint of *pain in the side of the chest*, *cough*, and *expectoration* or *dyspnœa*.

Pain in the side is caused mainly by affections of the pleura, and calls for an examination of the thorax. In differential diagnosis, it is not particularly valuable, since a mild pleurisy may accompany many other thoracic diseases.

Although *pain in the chest* may be called forth by some cardiac affection or by a muscular rheumatism, it demands a careful diagnostic examination. It may occur in any part of the chest, and may be described by the patient as a pressing or drawing pain.

A *cough* is almost invariably a sign of disease of the respiratory apparatus. It is reflex in its nature, and is caused by an irritation of the mucous membrane of the larynx, trachea, or bronchi by an accumulation of secretion. It can not arise from the alveoli of the lungs, although irritation or inflammation of the pleura may produce it. Occasionally a cough may be induced by an irritation of the pharynx, œsophagus, stomach, liver, or spleen.

The *kind* of cough may, at times, be of diagnostic significance.

It is important to notice the *number* of coughs that succeed each other in each attack. The so-called *hacking cough* is common in incipient phthisis, and may direct the physician's attention to the latent disease. The cough of *pertussis* is characteristic, and is seldom heard in other pulmonary diseases. It consists of a great number of coughs rapidly following each other and interrupted by deep, sighing, stridulous inspirations. A *dry* or *moist* cough, a *hard* or *loose* cough, give indications as to the amount and character of secretion and as to the progress of the disease. The *pitch* and *noise* made by a coughing patient will sometimes allow an estimate to be made of his general condition. A particularly loud, barking cough, for instance, is characteristic of inflammation of the larynx and trachea. A differential diagnosis, except in pertussis, can rarely be made from the mere symptom, cough; but the intensity of a pulmonary lesion, or its progress, may be recognized by the cough. Cardiac diseases sometimes engender a cough after a secondary pulmonary congestion has arisen.

Of the greatest importance in the diagnosis of diseases of the lungs is the *expectoration* or *sputum*. It must not be forgotten, however, that a catarrh of the nose or throat may be responsible for expectorated material, brought forth by *hawking*. Sputum which is expectorated by a cough comes from the larynx, the trachea, the bronchi, or from an ulcerating process in the lungs. Although the examination of the sputum usually concludes the examination of the respiratory organs, it is as well to take note of pathological peculiarities at once; as, for instance, bloody sputum or sputum of rusty-red or green color; balls of sputum or expectoration that sinks in water, etc.

INSPECTION OF THE THORAX

The inspection of the thorax gives information: 1. As to the *normal shape* or *deformity of the chest*.

Curvatures of the spine have a diagnostic importance, since they are etiological factors in some diseases of the lungs: through the compression of parts of the lungs and the compensatory emphysema of other parts; in the course of time this may be followed by dilatation of the right ventricle, and cyanosis and dyspnoea in consequence of the diminished circle of the pulmonary circulation.

Kyphosis is the term applied to a posterior spinal curvature, and is known as *gibbus* when it forms an acute angle. A forward curvature is known as *lordosis*, while a lateral curvature is called *scoliosis*. The most common form is the combination of *kyphosis* and *scoliosis*; i.e., a simultaneous lateral and posterior curvature.

Anomalies of the sternum have little diagnostic importance, as they do not produce pulmonary lesions. The xiphoid process may be pressed inward in the persons of men who, in their occupation, hold tools or implements against it, as shoemakers. A deep sinking in of the lower part of the sternum gives rise to the *funnel-shaped chest*; it is a congenital deformity, and is usually associated with other physical or mental abnormalities. A *pigeon breast* or *wedge-shaped breast* (*pectus carinatum*) is caused by rachitis by the compression of the costal cartilages. In this manner the sternum is pushed forward and assumes the shape of a wedge.

The angular eminence seen in many individuals between the manubrium and the body of the sternum is described as the *angle of Ludwig* (*angulus Ludovici*). The average length of the sternum in the adult is from 16 to 20 cm.

1. Of great diagnostic value is the circumference of the chest,—normal, *diminished or enlarged*; and it is important to note whether the enlargement or diminution exists on *one or both* sides.

a. A *bilateral increase* in the circumference of the thorax presents a characteristic picture; the chest is very broad, but seems shortened. It is known as the *barrel-shaped chest*,—and is pathognomonic for emphysema (*volumen pulmonum auctum*). Although the enlargement includes all the thoracic diameters, the antero-posterior is mainly deepened. The thorax is in a position of permanent inspiration.

The entrance of air or fluid into the pleural cavity of either side may produce an *enlargement of that half* of the thorax. The affected side *usually* lags behind in the respiratory movements. An unilateral enlargement occurs in pneumothorax and pleurisy with effusion, less often accompanying mediastinal tumors.

b. A *bilateral retraction* of the chest-walls is usually con-

genital. The thorax is long, flat, and narrow, the antero-posterior diameter is diminished, and the intercostal spaces are broad. This form of thorax has been described as a *paralytic thorax*, and generally arouses the suspicion of phthisis.

An *unilateral retraction* of the thoracic parietes, or better, perhaps, a sinking in of one side, is found after the absorption of pleuritic exudates and in the shrinking of tubercular lungs (*cirrhosis pulmonum*).

Measurements of the thorax. Dilatation or narrowing can usually be estimated quite distinctly with the naked eye; but it is sometimes necessary, in order to establish the circumference or measurements of the chest, to employ a tape-measure or a cyrtometer.

The mean average measurements of the thoracic circumference in a healthy adult man, estimated at the point of greatest expansion, are 82 cm. after deepest expiration and 90 cm. after deepest inspiration. This is, of course, only approximate, and varies largely in individuals. In general, it may be said that the circumference of the chest at the nipple after deepest expiration should equal one-half, at least, of the body's length. The right half of the chest may be from 0.5 to 2 cm. larger in a right-handed person than the left side, and *vice versa*. The thoracic diameters in women are somewhat less than the corresponding ones in men.

The sterno-vertebral diameter, measured with the cyrtometer, averages in adult men superiorly 16.5, inferiorly 19.2 cm. The lateral diameter at the level of the nipple (costal diameter), 26 cm. In women all these diameters are diminished.

3. The *frequency*, type, and rhythm of the *respiratory movements* may be appreciated by inspection.

The normal number of respirations varies from 16 to 20; in the new-born from 40 to 44. The normal relation between pulse and respiration is 1 to 3.5 or 1 to 4.

The lungs remain completely passive in respiration, merely following the motion of the ribs and diaphragm. In men the inspiratory expansion is due largely to the contraction of the diaphragm, and is known as the *costo-abdominal type* of breathing. The *costal type* is found in women in whom inspiration is principally accomplished by the elevation of the ribs by the intercostal and scaleni muscles.

An increased frequency and deepening of respiration (*dyspnœa*) is often caused by cardiac diseases and extensive abdominal distention.

Dyspnœa is to be referred to some pulmonary lesion when there is simultaneous cough and expectoration or pain in the side or other parts of the chest. It is a symptom of pneumonia, when accompanied by high fever and rubiginous sputum; it occurs in pleurisy and pneumothorax with impaired mobility and expansion of the affected side; it is seen in emphysema in connection with a barrel-shaped chest and diffuse bronchial râles over both lungs; it is observed, finally, in advanced phthisis (*habitus paralyticus*).

Dyspnœa which appears in a tubercular subject in an early stage of the disease is significant of some complication which must be recognized; as, empyema, pleurisy, pneumothorax, or miliary tuberculosis.

The kinds of dyspnœa. Inspiratory and expiratory dyspnœa are recognized. In inspiratory dyspnœa the accessory muscles of inspiration (the sterno-cleido-mastoid, scaleni, levatores costarum, pectorales major and minor, levatores scapulæ, rhomboidei, trapezii, and erectores trunci) are tetanically contracted. In extreme cases there is a retraction of the lower ribs and of the space just below the ensiform appendix. Stenosis of the larynx, trachea, or bronchi may call forth a high grade of inspiratory dyspnœa.

In expiratory dyspnœa, expiration is prolonged and difficult. The abdominal muscles, the quadratus lumborum, and the serratus posticus inferior act as accessory muscles. This variety of dyspnœa is found principally in emphysema and bronchial asthma.

The dyspnœa most commonly seen is a mixture of expiratory and inspiratory "air-hunger."

Asthma is that form of difficult breathing in which dyspnœa appears in attacks lasting at times for hours and followed by a longer or shorter period of quiet, natural respiration. Its most common form is *bronchial asthma*, probably of neurotic origin, which produces in apparently healthy individuals a temporary expiratory dyspnœa of the most intense kind. During such an attack the diaphragm is tetanically

contracted, the liver dulness is lower than normal, and diffuse, sibilant râles may be heard. At the end of the attack a small quantity of characteristic sputum is expectorated (see below). The pulse remains regular and strong during the attack. The prognosis after a single seizure is good so far as life is concerned; oft-repeated onsets lead to the development of emphysema.

In striking contrast to bronchial asthma are the dyspnoëic attacks in cardiac diseases (*cardiac asthma*). In these cases the pulse becomes small and irregular and the left ventricle is dilated. The prognosis must always be doubtful.

Nasal asthma is spoken of when attacks of bronchial asthma are evoked by reflex irritation of a pathological nasal mucous membrane (p. 108). *Dyspeptic asthma* is the name given to attacks of fear and anxiety in persons suffering from nervous dyspepsia. (In these instances one has usually to do with a neurasthenic.) *Uræmic asthma* consists of the attacks of dyspnoea occurring in chronic nephritis, which are probably in reality cardiac asthma. *Hay-fever* produces attacks of dyspnoea in otherwise healthy persons. It is probably caused by irritation of the nasal mucous membrane by the pollen of certain grasses.

SPIROMETRY

By inspection one may conclude as to the spirometry; that is, the determination of the quantity of air given off by the deepest possible expiration after the deepest possible inspiration (*vital capacity*). The vital capacity is *diminished* in all diseases of the respiratory organs.

The diagnostic value of this measure is unimportant, since there are no characteristic differences in the vital capacity among the various lung diseases.

On the other hand, however, spirometry is of real value in judging of the improvement or aggravation of a disease, particularly with reference to the influence of the therapeutic measures involved.

The vital capacity is measured by Hutchinson's spirometer. In healthy men it varies from 3000 to 4000 c.c., an average of 3600 c.c.; in healthy women it lies between 2000 and 3000, an average of 2500 c.c. The vital capacity increases with the bodily length, each centimetre of the latter representing about 22 c.c. of the former. In children and in the aged it is diminished.

Complementary air is that which may be drawn into the lungs by the deepest inspiration after an ordinary inspiration. It is about 1500 c.c.

Reserve air is that which may be expired by the deepest expiratory effort after ordinary expiration. It equals about 1500 c.c.

Breathing or *tidal air* is the air ordinarily changed by each act of respiration. It amounts to about 500 c.c.

Residual air is the air remaining in the lungs after the most violent expiratory effort. It is estimated at from 1600 to 2000 c.c.

After the thorough consideration of the conclusions reached by the inspection of the thorax and its movements, the *physical examination* of the respiratory organs is undertaken: the *percussion* and *auscultation* of the thorax.

MENSURATION

The topographical data which enhance the ease of the determination of the height and breadth of the chest are here given.

The *height* of the chest is *anteriorly* determined by the clavicle or by the supra- and infra-clavicular spaces. The external border of the latter includes the space of *Mohrenheim*. Below the clavicle the ribs are employed. The 2d rib is the starting-point in counting, since it can be easily recognized by its attachment to the sternum (angle of Ludwig). The nipple usually lies over the 4th rib or in the 4th intercostal space, usually about 10 cm. distant from the edge of the sternum. At the level of the xiphoid or ensiform process a distinct furrow usually traverses the thorax, which marks the site of insertion of the diaphragm (*Harrison's furrow*). The region beneath this furrow to the arch of the ribs is the *hypochondrium*. The determination of the height of the chest *posteriorly* is aided by using the spine of the scapula as a landmark; by this means the 7th cervical vertebra (*vertebra prominens*) is easily felt. The other guiding-point is the scapula, which covers the space from the 2d to the 7th or from the 3d to the 8th ribs and which is divided by

its spine into the supra- and infra-spinous fossæ. The space between the internal borders of the scapula is the inter-scapular space.

For the determination of the *width* of the thorax, the following imaginary lines, drawn parallel to the long axis of the body, are used:—

1. The *sternal line*, representing the borders of the sternum or the attachments of the ribs.
2. The *para-sternal line*, midway between the sternal line and the nipple.
3. The *mamillary line*, drawn through the nipple.
4. The *anterior axillary line*, drawn through the anterior border of the axilla (pectoralis major).
5. The *miffle axillary line* is drawn through the middle of the axilla.
6. The *posterior axillary line* is drawn through the posterior border of the axilla (latissimus dorsi).
7. The *scapular line*, drawn through the inferior angle of the scapula.

The linea costo-articularis is drawn from the claviculo-sternal articulation to the apex of the 11th rib.

Topography of the individual lobes of the lungs. The right lung has 3 lobes, the left but 2. The *right upper and right lower lobes* may be made out *posteriorly on the right side*. The border between them begins at the level of the 2d or 3d rib. This border is divided about 6 cm. above the angle of the scapula into two fissures which enclose the middle lobe. The superior fissure is directed almost horizontally forward and reaches the anterior border of the lung at the level of the 4th and 5th costal cartilages. The inferior fissure is directed perpendicularly downward and reaches the inferior border of the lung in the mamillary line. Hence we have: *right side, anteriorly, upper lobe* as far as the 3d rib; below that, *middle lobe*. On the *left side* posteriorly, upper lobe as on the right side. The border extends without bifurcation obliquely forward and ends in the mamillary line at the 6th rib. Hence we have: *left side, posteriorly, upper and lower lobes; anteriorly only the upper lobe* (and the heart).

PERCUSSION OF THE THORAX¹

By the percussion of different areas over the thorax, characteristic differences in sound may be appreciated according as the thoracic organs contain more or less air.

Percussion serves to determine:—

1. The borders of the air-containing lungs as differentiated from other organs (as the liver, the heart).
2. The amount of air contained in the lungs, which differs characteristically in diseases of these organs.

The *qualities of sound* obtained by percussion are:—

1. Clear or dull = loud and low.
2. High and low (pitch).
3. Tympanitic and non-tympanitic.

One obtains a clear (loud) non-tympanitic note over the lungs; a clear (loud) tympanitic note over gastric and intestinal areas; a dull note over the heart, the liver, the spleen. *High and low* are qualities referred only to tympanitic sounds (as over cavities).

Characteristic qualities of sound are: the *metallic sound* and the *cracked-pot sound* (*bruit de pot fêlé*).

It is essential for the beginner to practise percussion frequently on healthy persons, in order to drill his ear to the different qualities of the clear (normal) notes obtained over the lung. The normal note differs in intensity over the same lung, depending upon the muscular thickness and the deposit of fat. It is important to accustom one's self to compare the note obtained over analogous areas of the two halves of the body.

THE PERCUTORY DETERMINATION OF THE BORDERS OF THE LUNGS (FIG. 26)

Upper border: the determination of the upper borders of the lungs is important; because unilateral depression of the

¹ Percussion was discovered by Auenbrugger in Graz (1722-1809).

upper border is frequently the first sign of pulmonary tuberculosis. In healthy persons, the upper border extends from 3 to 4 cm. over the upper border of the clavicle; posteriorly it lies at the spinous process of the 7th vertebra. In healthy individuals, both borders are at the *same* level.

As the limit of the upper border is fixed, it may be simultaneously determined if the percussion note on one side is less clear than on the other. Differences in sound at the apices, in most instances point to tuberculosis (see below).

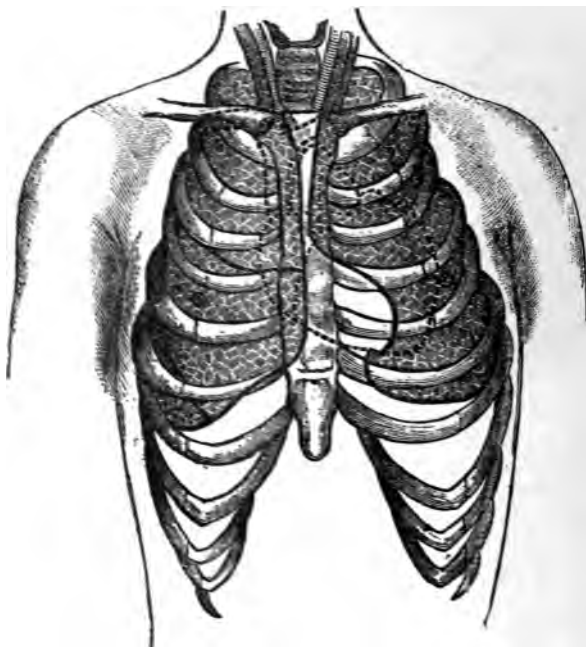


FIG. 26. — RELATIVE POSITIONS OF THORACIC VISCERA.

Lower border: while the determination of the upper borders of the lungs furnishes data for the diagnosis of phthisis, the establishment of the lower border aids in the diagnosis

of emphysema (*volumen pulmonum auctum*). In emphysema, the lower borders of the lungs are *lower* than in health.

The lower border of the lungs lies:—

On the *right* side, at the sternal border on the 6th rib; in the mamillary line, at the lower edge of the 6th or the upper border of the 7th rib; in the anterior axillary line, at the lower border of the 7th rib; in the scapular line, at the 9th rib; next to the vertebral column at the spinous process of the 11th dorsal vertebra.

On the *left* side, the lower border of the lung is difficult to make out, on account of the proximity of the stomach, the tympanitic sound of which merges gradually into the non-tympanitic sound of the lung.

It is customary to determine the lower border of the lung in the *right mamillary line*. In healthy persons, the relative liver dulness begins at the lower border of the 4th rib. The absolute liver dulness, *i.e.*, the lower border of the lung, begins at the lower border of the 6th or the upper border of the 7th rib. If one obtains a long clear note of some duration from the 7th to the 9th ribs, the diagnosis of emphysema is assured.

Temporary lowering of the lower border of the lung is found in attacks of bronchial asthma. The lower border of the lung is raised in meteorism, ascites, abdominal tumors; on one side only, indicates contracting pleurisy or atrophy of pulmonary tissue. The lower border of the lung on the right side is also pushed upward in pleurisy with effusion. This fact is demonstrated, of course, posteriorly; inferiorly on the right side.

Respiratory changes in the lower borders. In quiet, normal breathing, the lower borders sink about 1 cm. in the mamillary line; from 3 to 4 cm. in deep inspiration. The upper borders, in quiet respiration move upward about $\frac{1}{2}$, in deep inspiration about $1\frac{1}{2}$ cm. The respiratory displacement depends upon the position of the body. In the dorsal position, the anterior lower border of the lung moves 2 cm. lower than in the vertical position. The displacement in the axillary line is most marked, and may reach 10 cm. in deep inspiration with the patient in the side position.

Respiratory displacement is absent in extensive adhesions between the pulmonary and costal pleuræ, and is much diminished in emphysema.

DULNESS OVER PULMONARY AREAS

Dulness over the normal extent of the lungs may arise:—

1. When the lung *immediately* below the chest-wall is devoid of air; this area must have an extent of at least 4 cm. Pulmonary tissue may become deprived of air by *infiltration* and by *atelectasis*.

a. Infiltration is due to pneumonia, tuberculosis, abscess, more rarely gangrene, hæmorrhagic infarct, new growths, aneurysm.

b. Atelectasis arises from compression (pleuritic or pericarditic exudate and new growths) or from the resorption of air when the bronchi become occluded (by *tenacious* secretions or tumors and by syphilitic stenosis).

2. When *fluid* is exuded into the pleura, between the lung and the chest-wall (pleuritic exudate, hydrothorax); the quantity must be at least 400 c.c. Dulness is evoked, also, by thickening of adherent layers of pleura (*adhesions*).

Dulness over the upper lobes (apex) (with good resonance behind, below) usually indicates pulmonary phthisis, more rarely pneumonia, gangrene, or new growths. Dulness over the lower lobes (posteriorly, below) usually indicates pneumonia or pleurisy. The differential diagnosis is made by auscultation. Tubercular infiltration of the lower lobes is usually secondary to extensive tubercular invasion of the upper lobes. In rare cases, dulness of the lower lobes is to be referred to gangrene, infarct, or tumors. In bronchitis and miliary tuberculosis, there is *no* dulness present.

In protracted diseases, with the patient constantly in the dorsal position, serous infiltrations causing dulness may appear in the lower portions of both lungs (*hypostatic congestion*).

TYMPANITIC RESONANCE IN THE AREA OF THE THORAX

In the healthy thorax, a loud tympanitic note is obtained only over the stomach (*semilunar* space, p. 83), and over the parts of the left lung immediately adjacent to it. These parts are thin enough to allow the tympanitic note of the stomach to be evoked by percussion.

The lower portion of liver dulness may be concealed by the tympanitic note due to tympanites.

A tympanitic percussion note in other parts of the thorax arises:—

1. In *cavities* in the parenchyma of the lung; the cavity must have at least the size of a large walnut, must lie near the chest-wall or must be connected with it by infiltrated tissue. Such cavities are present in phthisis, less often in bronchiectasis and gangrene.

2. In a collection of air in the pleural sac (*pneumothorax*), but only while the air is not under too great tension. Otherwise the note is deep and loud, but *not* tympanitic.

3. In complete infiltration of large pulmonary areas, by which there is a good conduction of sound between the broncho-tracheal column of air and the chest-wall; for instance, in tubercular infiltration of an entire upper lobe, and in the second stage of pneumonia (dulness with tympanitic sound).

4. In diminished tension (relaxation, atony) of the pulmonary tissue: over pleuritic and pericarditic exudate and pneumonic infiltration; frequently in the first and third stages of croupous pneumonia and in oedema of the lungs.

All conditions in which a tympanitic note is heard have this in common: that a column of air *above* is brought into vibration (without vibration of the tissues of the lung).

In the cases of cavities and *pneumothorax*, the column of air in the space is percussed directly: in the instance of extensive infiltrations, one percusses the normal cavities (broncho-tracheal

column of air through the thickened tissues. Pulmonary tissue lying above exudates is robbed of its elasticity and vibratory power, and hence, in percussion, the air only contained in the lung vibrates.

A metallic amphoric note differs from a tympanitic note by its higher pitch and by its longer duration. In addition to the fundamental note, there are overtones to be heard, which slowly disappear. They may be compared to a metallic echo.

Amphoric resonance may be elicited over the thorax:—

1. In the presence of cavities with homogeneous walls, which must have the size of a man's fist at least (6 cm. diameter).

2. In pneumothorax, when the air present is under a certain pressure, not too great.

In order to appreciate amphoric resonance more distinctly, percussion by means of the *hammer* and *pleximeter* may be employed (mediate percussion). While performing auscultation over the cavity, the pleximeter, placed next to the stethoscope, is lightly hit by the hammer. By this means a clear, metallic note is elicited.

The *cracked-pot sound* (*bruit de pot fêlé*) is brought out by a short, sharp percussion. It is best obtained with the patient's mouth open, over a superficial cavity which communicates with a bronchus by a narrow opening. The sound may resemble that of the *rattling of coins*. Caution must be observed in estimating the value of the cracked-pot sound, as it may appear in healthy persons, especially children, in speaking and singing; and moreover, is sometimes found in tissues relaxed and infiltrated (pleurisy and pneumonia).

CHANGES OF SOUND AND SYMPTOMS OF CAVITIES

A tympanitic note is *high* or *low* according to the length of the column of air which produces it, and the breadth of the opening by which it reaches the external air. The

shorter the column of air and the wider the opening, the higher the note. By having a patient open his mouth, the opening, communicating with a cavity which is freely connected with a large bronchus, may be enlarged; by changing the position of a patient, the diameters of a purulent cavity, with unequal diameters, may be changed. By thus changing the physical conditions, high and deep notes may be artificially produced. This is known as sound mutation or change.

1. The *change of sound* known as *Wintrich's* consists of the change produced in a tympanitic note by the opening and closing of the mouth — higher in the former, lower in the latter case. Found in lung cavities and *pneumothorax* when these communicate freely with a bronchus. Rarely in pneumonia and over pleuritic exudates.

One can imitate this phenomenon by percussing the larynx with alternate opening and closing of the mouth.

An *interrupted Wintrich's sound change* is the designation given to the disappearance of the phenomenon when the patient sits up, while it was easily obtained when he was lying down, and *vice versa*. It is caused by the occlusion of the bronchus, which in a certain position of the body freely opens into the cavity.

2. *Gerhardt's change of sound*: when the patient sits up, the tympanitic note is deeper than when he is lying. This phenomenon appears in oval cavities which are, in part, filled with fluid; the note has the lowest pitch when the longest diameter is horizontal. When this diameter is diminished by change of position, the note becomes clearer. (If the note becomes higher when the patient sits up, the diagnosis of a cavity is not certain.) This phenomenon may be produced by percussing a partly filled bottle.

3. *Respiratory change of sound*: in very deep inspirations, the tympanitic note over cavities sometimes rises in pitch, probably by the increased width of the glottis, which represents an increase in the size of the cavity.

4. *Biermer's change of sound*: percussion over a *pneumothorax* containing fluid, the percussion note is deeper in the recumbent than in the sitting posture, because the long diameter of the air-containing pleura becomes increased, in the lying position, at the expense of the long diameter of the exudate, which sinks from the diaphragm upon the posterior chest-wall.

AUSCULTATION OF THE THORAX¹

By means of auscultation one appreciates: (1) the respiratory murmur; (2) râles and friction sounds.

THE RESPIRATORY MURMUR

Vesicular, bronchial, amphoric, and broncho-vesicular breathings are distinguished.

Vesicular breathing (*cell-breathing*) is found over the entire healthy lung. It is principally audible on inspiration; of a shuffling character, and is not heard on expiration, or if heard, is short and uncertain, and is rarely audible as a vesicular murmur.

Vesicular breathing may be easily imitated by placing the lips and teeth as though one were to pronounce a soft *f* and then taking a deep *inspiration*. Vesicular respiration arises in the trachea and the large bronchi, and is therefore really bronchial breathing, which attains its peculiar shuffling character through being conducted into the small bronchi and the alveoli of the lung.

Clear, soft vesicular breathing without râles is a positive sign that the auscultated area of lung is healthy.

Diminished intensity of vesicular respiration is found in emphysema, since, in consequence of the distention of the lung tissues, only a small quantity of air enters the alveoli; in pleurisy, because the fluid between the chest-wall and the lung is a poor conductor of sound; in instances of adherent and thickened pleuræ, because the lung enveloped by them can not unfold itself. Over very large pleuritic exudation, the respiratory murmur is entirely absent.

An *increased intensity of vesicular breathing* is normal in children: *puerile respiration*. It appears in swelling and stenosis of the bronchi and in bronchitis (because the current of air is inspired with greater force to overcome the obstructions present).

¹ Auscultation was discovered by the Parisian clinician Lænnec (1781-1826).

An *increased intensity of vesicular expiration* and a prolongation of it arises when there are obstructions to the exit of air from the alveoli because of narrowing of the smaller bronchi, as in bronchitis and bronchial asthma. Prolonged expiration increased in intensity over *the apex of the lung* is an early sign of phthisis.

Interrupted or jerky respiration is vesicular breathing in which inspiration is accomplished with several intermissions. It may appear in healthy persons who breathe slowly and irregularly; but it is often found at the apex as an early sign of tuberculosis. It must not be given too high a value, and is to be considered only in connection with other symptoms.

Systolic vesicular breathing refers to the increased respiratory murmur often heard in the neighborhood of the heart during its systole. It has no diagnostic significance.

Bronchial breathing (*cavernous respiration*) is found in health over the larynx, the trachea, and the inter-scapular space. It is of a puffing character, principally audible on *expiration*; on inspiration it is usually heard shorter and weaker.

Bronchial breathing may be imitated by putting the mouth in the position of pronouncing the soft German *ch* and slowly *expiring*. Bronchial breathing arises through the rotatory motion imparted to the inspired air as it passes through the rima glottidis, which is in turn continued in the broncho-tracheal column of air.

Bronchial breathing is produced by analogous conditions to those which call forth a tympanitic percutory note:—

1. In large cavities, but the conducting bronchus must be free.

2. When the lung is so *consolidated* that the bronchial respiratory murmurs of the large bronchi are conducted unchanged to the chest-wall:

- (a) in infiltration by pneumonia or tuberculosis, less often, gangrene;
- (b) in compression, particularly above exudates into the pleura.

Dulness and bronchial breathing over the lower lobe, with an uninvolved upper lobe, usually denotes pneumonic infiltration. Dulness and bronchial breathing over the apex, with a normal lower lobe, usually indicates tubercular infiltration. But it must not be forgotten that these symptoms may point to an upper-lobe pneumonia, a lower-lobe tuberculosis, gangrenous infiltration, pleuritic thickening, or compression due to tumors or aneurysms.

A loud, tympanitic note and bronchial breathing denote a cavity; a loud deep note with no respiratory murmur, a closed pneumothorax; dulness with no respiratory murmur, a pleuritic effusion.

Metamorphic respiration is the name given to an uncommon respiratory murmur which begins as vesicular and becomes bronchial. It is heard, when at all, over cavities, but is not at all characteristic.

Amphoric respiration is never heard over the healthy chest; it is a whistling respiratory sound with a metallic echo, produced under the same conditions as the metallic percussion note. It is pathognomonic for cavities with walls of uniform density of a diameter of at least 6 cm., and for an *open pneumothorax*. Over a closed pneumothorax no respiratory murmur is heard.

Amphoric respiration may be imitated by blowing over the top of a large bottle.

Broncho-vesicular breathing is breathing which is neither distinctly bronchial nor distinctly vesicular. One should be very cautious in diagnosing this variety of respiratory murmur, and should try, by careful auscultation, and by having the patient take deep inspirations, to give the murmur a vesicular or a bronchial character.

Broncho-vesicular respiration is found in healthy persons, on superficial breathing, in the supra- and infra-spinous regions. In pathological conditions it is frequently heard without allowing any diagnostic conclusion to be drawn from

it. When broncho-vesicular breathing is *permanently* heard at one apex, it may be regarded as a sign of beginning tuberculosis.

RÂLES AND FRICTION SOUNDS

The adventitious respiratory sounds are always signs of a diseased condition of the mucous membrane of the lung, or of a collection of secretions, or of pus. Râles are known as *dry* and *moist*.

Dry râles are caused by the passage of the inspired air through a narrowed bronchus or one covered with a tenacious secretion. They may be of a *purring* or *sonorous* (*rhonchi sonori*), or of a *whistling* or *sibilant* (*rhonchi sibilantes*) character. They are pathognomonic for bronchitis.

Moist râles are heard when there is a collection of secretions which conduct the inspired air, or when bubbles containing air are ruptured, or when closed alveoli or bronchioles are forced open.

Moist râles may be *plentiful* or *scattered*, *fine* or *coarse*; they may be *ringing* or *not*, *metallic* or *not*.

The coarseness of râles depends upon the size of the cavity in which they arise; fine râles are usually derived from the small bronchi in a beginning infiltration; coarse râles in large bronchi and cavities.

Crepitant râles (bullous râles) take their origin in the forcing open of previously closed alveoli, and are a subdivision of fine râles. They may be imitated by rubbing the hair between the fingers. Crepitant râles are heard in the 1st and 3d stages of pneumonia, in œdema of the lungs, in miliary tuberculosis; sometimes, in the apices and the areas of lung immediately beneath them, in *healthy* organs during the first few inspirations after partial atelectasis.

Ringling râles are often heard under the same conditions as bronchial breathing (in cavities, in large infiltrations which act as sound conductors, in compression).

Metallic, ringing râles are often heard in connection with a metallic percussion note and amphoric breathing.

A single metallic râle is known as tinkling (*tintement métallique*, the sound of falling drops of water). Heard in pneumothorax.

Succussion (*succussio Hippocratis*) is a metallic splashing heard at some distance from a patient when the upper part of his body is energetically shaken from side to side. It is pathognomonic for the simultaneous presence of air and fluid in the pleural sac (sero- or pyo-pneumothorax).

Succussion may also be heard over a large stomach after the ingestion of fluids. It speaks in this instance in favor of gastric dilatation, but does not prove it.

Friction sounds (pleuritic) are heard when two adjacent layers of pleura, rubbing against each other in the respiratory act, become roughened by deposits of fibrin. They are pathognomonic for pleurisy, are most frequent in dry pleurisy, and in large effusions are heard usually in the stage of resorption. Friction sounds are absent in effusions with stasis (hydrothorax) and when adhesions between the layers of the pleura exist.

The distinction between friction sounds and dry râles is sometimes difficult to make. It may be remarked that râles are changed by coughing, friction sounds are not; that friction sounds are increased in intensity by the pressure of the stethoscope, and that under these circumstances a complaint of pain is made; that friction sounds are appreciable to the hand.

They may be compared to the creaking of new soles and to the rustling of a tense sail.

PECTORAL FREMITUS

Pectoral or vocal fremitus is appreciated by placing the hands symmetrically on the two sides of the chest while the patient speaks or counts in a deep voice. In healthy persons one feels a distinct vibration of the chest-wall produced by the conduction of the voice.

Pectoral fremitus is *increased* in pneumonia, above pleuritic effusions, in cavities with thickened walls. (Infiltrated and compressed tissues are good conductors of sound; in cavities the sound of the voice is intensified by reflection from the walls.)

Vocal fremitus is *diminished* or *absent* : —

- a. When the pleural sac is filled with fluid or with air (*pleurisy* and *pneumothorax*). Over pleural adhesions, vocal fremitus is frequently well preserved.
- b. When the large bronchi are occluded by tumors or stenosis.

The diminished intensity or absence of vocal fremitus is of diagnostic value only when the voice is strong and deep. A weak voice produces a weak fremitus. In very stout people, it can not be well felt.

Auscultation of the voice. The auscultation of a healthy chest during talking gives only an indistinct humming. This is *diminished* under the same conditions as vocal fremitus. The voice seems to be *intensified* when the waves of sound travel through good conductors, that is, through infiltrated or compressed tissues, or through cavities with thickened walls. Vocal auscultation is therefore bounded by the same limitations as vocal fremitus and bronchial respiration. An increased auscultatory intensity of the voice is called *bronchophony*; very much heightened intensity, *pectoriloquy*.

Ægophony is the name given to a peculiar sound of the voice, possessing a tremor and sounding like the bleating of a goat, which is often heard at the upper limit of an extensive pleuritic effusion.

EXAMINATION OF THE SPUTUM

The examination of expectorated material coughed up is indispensable for the diagnosis of diseases of the lungs. The examination begins with a simple inspection of the sputum (macroscopic examination) which is followed in case of necessity by a microscopic examination.

Every sputum may be placed in a group according to its main constituents: *mucous, purulent, serous, fibrinous, bloody* sputum; or it may represent a mixed form, as *mucopurulent, bloody-serous*, etc.

1. *Pure mucous sputum* (tenacious, glossy, sticking to the bottom of the glass) is characteristic of a beginning bronchitis. As bronchitis may be the forerunner of tuberculosis, a sputum consisting of pure mucus must be given cautious diagnostic judgment. The expectoration of the nose and pharynx is often purely mucous.

2. *Pure purulent sputum* (thick, confluent pus, not foamy) is found almost solely in perforation of purulent foci: empyema, abscess of the lungs or of the neighboring organs; also in bronchorrhœa.

3. *Muco-purulent sputum* is found most frequently, and has no characteristic points for differential diagnosis. Is seen in severe bronchitis as well as in phthisis pulmonum. In bronchitis pus and mucus are often intimately mixed; in phthisis it frequently consists of single balls which have a gnawed appearance, are surrounded by mucus, and sink to the bottom. This kind of sputum (nummulous, *globosum et fundum petens*) is mostly characteristic of tubercular cavities, but occasionally appears in bronchorrhœa.

Profuse mucopurulent sputum often forms *three layers* on standing: the lowest, pus, then serous fluid, on top foamy mucus. Found most often in bronchiectasis and cavities, but is not pathognomonic.

4. *Serous sputum*, thin, fluid, usually stained a light red, is pathognomonic for œdema of the lungs; its appearance is usually a bad prognostic sign, usually a sign of early death (stertor).

5. *Pure bloody sputum* (*hæmoptysis*) is expectorated when through some ulcerative process a pulmonary blood-vessel becomes eroded, or when in the pulmonary circulation or in isolated areas (embolism), stasis of high degree is present.

Differential diagnosis as to hæmatemesis (p. 79). Hæmoptysis appears:—

- a. Principally in *tubercular phthisis*; sometimes in the first stage (initial hæmoptysis) or in any stage of the subsequent course of the disease; the amount of the coughed-up blood varies from 1 to 2 teaspoons to $\frac{1}{2}$ litre or more. The proof of the presence of other signs of tuberculosis is required to make a positive diagnosis.
- b. Less often in abscess of the lung or gangrene.
- c. In extensive stasis in the pulmonary circulation, especially in mitral lesions.
- d. In hæmorrhagic infarcts of the lung; the source of the embolus must be proven (venous thrombosis, thrombosis in the right ventricle); when possible, the area of dulness of the infarct, or râles, must be elicited.
- e. In aneurysm of the aorta which may lead to profuse, often fatal, pulmonary hæmorrhage.
- f. In very rare instances, when a dilated vein (varix) of one of the large bronchi bursts in an otherwise healthy person. This diagnosis is justified only when the other causes are excluded.

An hæmoptysis which appears at the time of suppressed menses in a young girl may be regarded as beneficial (vicarious hæmoptysis). One must not neglect the examination for tuberculosis, however.

6. *Bloody-mucous sputum* (like raspberry jelly) is occasionally seen in carcinoma of the lung; when it is more bloody than mucous, sometimes stained yellowish-brown, in the first stage of pneumonia and in hæmorrhagic infarcts.

Saliva stained with blood is often expectorated by hysterical patients, and may lead to error.

Fibrin in quite large quantities is seen in fibrinous bronchitis and pneumonia; if one shakes up the sputum with

water in a test-tube, branches of fibrin like those of a tree appear (*bronchial casts*).

In order to decide whether sputum contains fibrin, *Ehrlich's* three-color mixture may be employed. This tri-acid mixture contains acid fuchsin, methyl-green, orange-G. The acid fuchsin is absorbed by the pure albumin present, while nuclein (the albumin of the nucleus) and mucin are stained with the basic methyl-green. If a specimen of sputum is shaken up with this mixture, pneumonic expectoration will be stained red because of the fibrin it contains, the sputum of bronchitis will be a bluish-green because of the mucus and leucocytes it holds. This test may be employed microscopically on dry specimens.

The smell and color of the sputum are next to be observed. Most sputa have an insipid, sweetish smell. A mouldy odor may accompany material arising from the mouth, the teeth, the nose, and the pharynx. A *sickly, foul odor* is a sign of purulent bronchitis or gangrene of the lung or ruptured putrid abscesses (see below).

A foul odor may come from the oesophagus or nose, which must always be carefully examined as possible sources of putrid sputum.

The color is usually a yellowish-green. Sputa of other colors have important bearings: *red color* (see *bloody sputa*).

Rubiginous (rusty) *sputum*, pathognomonic for pneumonia.

Ochre-yellow sputum, pathognomonic for the perforation of foci of liver disease (echinococci, abscesses from biliary calculi, necrotic liver tissue). The color comes from the large quantity of bilirubin.

A similar color is caused by the activity of bacteria: *sputum* of the color of the *yolk of an egg*. The color becomes more intense when exposed to the air, and if a small part of the sputum is transferred to clear sputum, the latter assumes the yellow color in a short time.

Grass-green sputum, characteristic of a slow resolution of a pneumonia, usually indicates a transition to tuberculosis; also seen in pneumonia accompanied by icterus.

The green color may also be caused by bacterial life. This form may be recognized by its imparting its color to clear sputum.

The differently colored sputa which acquire their color from external sources are without diagnostic significance: *blue sputa* in workmen in chemical factories, *red* and *yellow* in workers at the forge, *black* in workers in coal.

Lastly, the **amount** of sputum in 24 hours must be noted. For diagnostic purposes it may be important when there is a very great quantity of secretion (purulent bronchitis, bronchiectasis, tuberculous cavities, perforating empyema, etc.). In many cases the intensity of the pathological process may be estimated by the amount of sputum.

MICROSCOPIC EXAMINATION

A microscopic examination of the sputum must be made in all cases in which the examination of the thorax and the inspection of the sputum have not led to a positive diagnosis (for example, when there is a suspicion of tuberculosis, in the presence of blood-stained, mucous sputum and ill-smelling sputum, etc.).

Important elements are: elastic fibres, fragments of lung tissue, tubercle bacilli, heart-disease cells,¹ eosinophile cells, Leyden's asthma crystals, and Curschmann's spirals. These are some elements which help to secure the diagnosis made: crystals of the fatty acids, hæmatoidin crystals, bronchial casts, etc.

Elements which lack particular diagnostic importance are (Fig. 27): *White blood-cells*, present in large quantity in every sputum, often degenerated, often fatty.

Pavement epithelium comes from the mouth and the true vocal cords.

Cylindrical epithelium is not often found; it comes from the nose, the upper pharynx, the lower part of the larynx, and the bronchi.

¹ We have translated the word "Herzfehlerzellen" as above. — THE TRANSLATORS.

The most common cells are large round or round with a nucleus which sometimes is indistinct and are usually filled with particles of food. It will be the most typical leukocytes. Although the presence of a small number of these cells is not characteristic of tuberculosis, the presence of a large quantity of leukocytes will almost always awaken a suspicion of the disease.

Small red blood cells are without significance. They are present in abundance in bloody sputum.

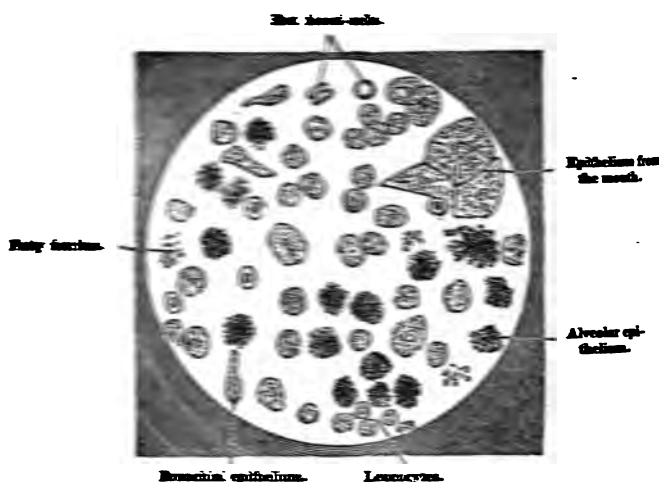


FIG. 2. — MOWING SPUTUM IN CHRONIC BRONCHITIS, CONTAINING NO PATHOLOGICAL ELEMENTS.

Sarcina pulmonum is seen now and then. It has no diagnostic importance.

The ordinary bacteria are present in profusion in every sputum, particularly in old sputum, and have no diagnostic value. *Leptothrix* (stained blue with Lugol's solution) are present in gangrene; they also appear in the follicles of the tonsils in the healthy. Lugol's solution contains iodine, 1; iodide of potassium, 2; distilled water, 100 parts.

CHARACTERISTIC ELEMENTS

Elastic fibres, in all destructive processes: tuberculosis, abscess, gangrene. In gangrene not so many fibres are found, for there is a ferment in the sputum which dissolves them. Abscess is rare and is to be diagnosticated from other symptoms (see below), so that, as a rule, the presence of elastic fibres speaks in favor of tuberculosis.

To find elastic fibres, a cheesy particle may be selected from the sputum with a curved forceps. The sputum should be poured on a black plate to render the selection easier. Low powers of the microscope will answer for the preliminary examination. The sputum may be previously boiled in a test-tube with an equal quantity of a 10 per cent. potassium hydrate solution and allowed to precipitate. The sediment is then examined microscopically.

Fragments of lung tissue appear as small black particles, visible to the naked eye, in abscess and gangrene. In foul-smelling sputum their presence speaks in favor of gangrene rather than fetid bronchitis.

Heart-disease-cells are found in the sputum in all diseases in which there is chronic pulmonary congestion, as in mitral disease and in all cases of dilatation of the left ventricle. These conditions frequently superinduce cardiac asthma and secondary emphysema. The heart-disease-cells are desquamated alveolar epithelium containing granular, brownish-yellow pigment.

The source of this pigment is undoubtedly the blood, and may be proven by the hæmosiderin reaction: to a fresh specimen diluted hydrochloric acid and the yellow potassium ferrocyanide (5 per cent. solution) are added. The pigment granules take on a blue color from the iron they contain.

Eosinophile cells appear in the sputum usually in combination with Leyden's asthma crystals. They are large cells with very fine, colorless granulations which stain a beautiful red with eosin. Their presence in abundance seems to

bear some relation to attacks of asthma; but nothing definite can be said as to their significance.

The staining of these cells in dry specimens may be best accomplished by the following solution (Dr. Bein): concentrated aqueous

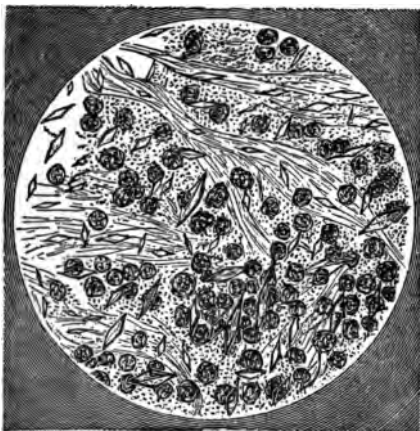


FIG. 28. — ASTHMA CRYSTALS.

solution of methylene blue 50; absolute alcohol and distilled water, of each 24. To this solution is added a piece of eosin the size of a bean. In this solution leucocytes and bacteria are stained blue; the eosinophile granulations, red.

Leyden's asthma crystals (Fig. 28), strongly refractive, octahedral figures, are found almost solely in bronchial asthma. Macroscopically,

they appear in yellow, sausage-shaped particles. Chemically, they consist of diethylendiamin.

Curschmann's spirals (Fig. 29), also found in the sputum of asthma but seen much less frequently, may be recognized with the naked eye, better with a magnifying glass, as fine thread-like bodies, often contained in sago-like particles of mucus. They are conglomerations of mucus wound in the manner of a corkscrew, with a light central line.

Fibrin may often be appreciated macroscopically. Shaken with water, it may be recognized by its forming bronchial casts shaped like the branches of a tree. Microscopically, it is known by its brightness, its fine streaking, and its homogeneity (staining reaction, p. 144). Fibrin is found in asthma, fibrinous bronchitis, and pneumonia.

Crystals of the fatty acids usually present themselves as curved,

colorless needles, frequently in bunches; are easily distinguishable from tyrosin and other crystals by melting when warmed. Are present in gangrene and fetid bronchitis, usually contained in yellow, ill-smelling plugs the size of a pinhead (*Dittrich's plugs*).

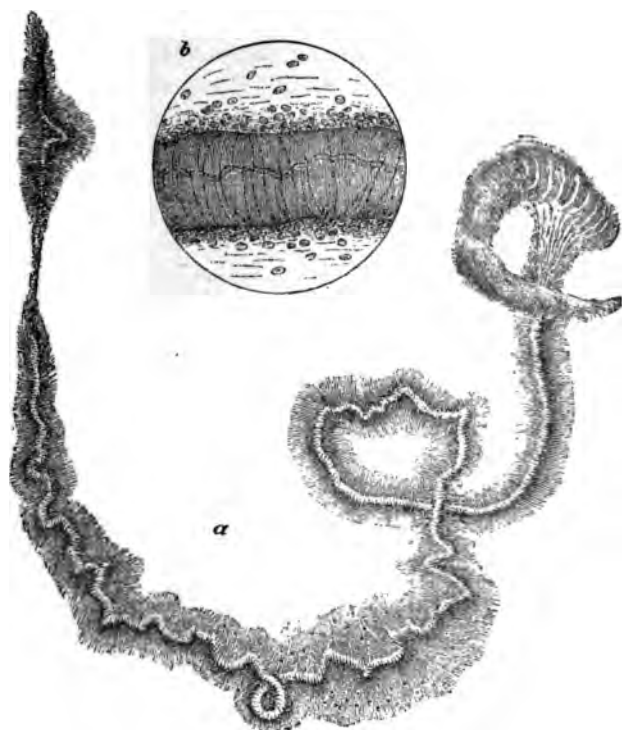


FIG. 29. — CURSCHMANN'S SPIRALS.

a: magnified 80 times. *b*: a piece of *a*, magnified 300 times.

Hæmatoidin crystals are found in old hæmorrhages, especially in abscess and perforating lesions of the liver (ochre-yellow sputum). They occur in brownish-yellow groups, as isolated needles, rhombi, and flakes.

Cholesterin crystals are six-angled, notched plates seldom found in old, purulent sputum (abscess and cavities).

Tyrosin crystals are bunches of needles, in old pus, particularly in the drying pus of perforating empyema.

Small cysts of echinococcus or its hooks are rarely present in the sputum, and when they are, denote echinococcus of the lung or perforation from some neighboring organ (see Chap. XII.).

The *cocci of pneumonia* (see Chap. XII.) are present in profusion in every case of genuine fibrinous pneumonia. A simple glance does not suffice, however, to base a diagnosis on, since it is difficult to distinguish them from harmless fungi. They may be recognized with certainty if one emulsifies a quantity of sputum with sterilized water and injects this subcutaneously into a rabbit. If the sputum contain virulent pneumococci, the rabbit will die of septicæmia in from 1 to 3 days, and its blood and the fluid from its enlarged spleen will contain innumerable diplococci. The expectorated material employed must be free from saliva and bronchial mucus and must be positively from the *lungs*; for pneumococci are present in the *mouths of healthy persons* and are therefore also called the cocci of sputum septicæmia.

To cleanse the pulmonary sputum, as above mentioned, it is received in a sterilized Petri's dish; it is washed with sterilized water, transferred to another similar dish with sterilized forceps, is again washed in sterile water, etc. After this process is repeated five or six times, it may be safely assumed that the sputum is free from contamination, and may be employed for purposes of culture or inoculation (*Koch's washing method*).

Influenza bacilli (Chap. XII.) are found abundantly in the bronchitis and pneumonia of influenza. The microscopic examination does not always answer, and it may be necessary to make a culture.

Tubercle bacilli. Their demonstration is the keystone of the diagnosis of tubercular phthisis of the lungs. In doubtful cases, especially in the early stages, the proof of their presence is of the highest importance. The finding

of tubercle bacilli in the sputum proves with certainty the diagnosis of tuberculosis. A negative result, on the other hand, does not speak absolutely against the diagnosis. For the technic of staining, see Chap. XII. If difficulties present themselves, the method of *Biedert* may offer some advantages. A teaspoonful of sputum, with 3 teaspoonfuls of water and 15 drops of potassium hydrate, are boiled for 2 hours over a sand-bath. The greater part of the pus is dissolved. In the sediment remaining, even a few bacilli may be stained easily and with certainty.

Anthrax bacilli and the club-like ends of *actinomyces* have been occasionally found in the sputum as proof of the existing disease. So have *chains of aspergillus* and the *fungus of thrush* in the sputum of pneumonomycosis.

SYMPTOMS OF THE DISEASES OF THE LUNGS

Bronchitis. — Cough and expectoration, frequently pain in the chest, usually no marked emaciation. Physical examination: *no* dulness, vesicular breathing often accompanied by prolonged sharpened expiration, diffuse dry râles. Sputum, in acute cases, glassy, mucous, clinging tenaciously to the bottom of the containing vessel; in chronic cases, muco-purulent, with no characteristic elements. Signs of bronchitis in *one* apex only justify the suspicion of beginning tuberculosis (*catarrhus unius lateris non est catarrhus*). It is worthy of note that signs of acute bronchitis arise secondarily: in cardiac disease (bronchitis of stasis), and in the acute infectious diseases, as in typhoid fever. Prognosis of simple acute bronchitis, with proper treatment, good. Chronic bronchitis leads to emphysema and dilatation of the right heart, and runs its course, frequently, with attacks of bronchial asthma.

Chronic bronchitis may lead, through retention of the secretions, to sac-like dilatations of the smaller air-passages

(*bronchiectasis*). Small bronchiectases usually escape diagnosis. In large bronchiectases, there may be a marked stagnation of pus, which undergoes putrefaction (see fetid bronchitis). The sputum, in such cases, is expectorated in the morning frequently by mouthfuls, and arranges itself in three layers. Large bronchiectases give the physical signs of cavities; they usually occupy the lower lobes, and may be concealed by the corresponding compression of the tissues of the lungs.

The etiological irritation which is the cause of bronchitis (dust, etc.) may also evoke interstitial inflammation in the lung, which may lead to formation of connective tissue, and to contraction (interstitial fibrous pneumonia, cirrhosis of the lung, pneumoconiosis). Bronchitis may also, in this way, be responsible for areas of dullness. It must not be forgotten that tubercle bacilli usually find their way into lungs saturated with dust and irritated by it; and in practice it has this significance,—that bronchitis with dullness denotes tuberculosis. The final decision will rest upon the finding of tubercle bacilli.

Emphysema (*volumen pulmonum auctum*).—Shortness of breath and cyanosis, usually cough and expectoration. Barrel-shaped chest. Often dilated veins on both sides of the chest. Lower border of the lung lower than usual; absence or diminution of cardiac dullness. Vesicular breathing diminished in intensity. Frequently, dry râles.

The prognosis depends upon the degree of preservation of the respiratory motility of the lungs; in very advanced cases, the limit of the expiratory position of the lower border of the lungs is from $\frac{1}{2}$ to 1 cm. from their most extended inspiratory position. Some observers reserve the name *emphysema* for those serious cases prognostically, in which the alveoli undergo necrosis, because of the pressure prevailing in them, thus producing a typical (Laënnec's) emphysema, in which air, to a certain extent, penetrates the tissues. If one employs the word *emphysema* in this limited sense, the phrase *volumen pulmonum auctum* would serve well for all those cases of moderate and milder forms which represent clinically a completer conservation of respiratory normality.

Pneumonia (*genuine, croupous, lobar*). — Sudden beginning with chill, pain in the side of the chest, cough. High continuous fever. Rubiginous sputum. Physical signs, — 1st stage (engorgement): tympanitic, slightly dull note over the infiltrated (lower) lobe; crepitant râles; in the 2d stage (hepatization), complete dullness; bronchial breathing. Increased vocal fremitus. 3d stage (resorption or resolution): gradual clearing up of the dullness, the bronchial breathing gradually becomes vesicular; fine and coarse râles may be heard. Prognosis: usually favorable; crisis between the 3d and 11th days, resolution of the exudate (disappearance of dullness) in 1 to 4 weeks. In weakened persons, the resolution may require months (as much as $1\frac{1}{2}$ years) (delayed resolution). Fever of remittent type lasting longer than 11 days points to complication or sequelæ, especially pleurisy (empyema); rare issues are carnification (formation of granulation tissue in the exudate with subsequent connective tissue formation and contraction), cheesy degeneration, abscess, gangrene. Prognosis for healthy young persons usually good; in individual cases the prognosis depends particularly upon the condition of the heart (pulse) and the involvement of the mental faculties. Prognosis grave in drunkards, aged people, cardiac cases, and persons with kypho-scoliosis.

Broncho-pneumonia (*secondary, catarrhal, lobular*). — Follows bronchitis, usually in acute infectious diseases: moderate dullness often with a tympanitic sound, with bronchial breathing and moist râles, over circumscribed areas most frequently involving both lower lobes. Remittent fever of long continuance, sputum usually muco-purulent, colorless. Appears chiefly in children and the aged, frequently in stuporous patients who aspirate particles of food (aspiration pneumonia).

The prognosis of a broncho-pneumonia is always grave; recovery is possible, though most frequently death occurs as a result of exhaustion, tuberculosis, or gangrene.

Dry pleurisy (*pleuritis sicca*) is diagnosticated by the presence of localized *friction sounds*, which are equally loud on inspiration and expiration, and may frequently be felt. Pain and usually fever. Not rarely caused by exposure to cold. Often present in tuberculosis.

Pleurisy with effusion (*pleuritis exsudativa*). — Begins suddenly with a chill and pain in the side, or the pain may come on gradually; frequently low degree of *dyspnoea*. Fever irregular, remittent. Absolute dulness (flatness) below posteriorly, respiratory murmur and pectoral fremitus diminished or absent. Above the border of dulness, on account of the compression of the lung, often a tympanitic, slightly dulled percussion note, bronchial breathing, very fine râles (atelectasis). Apex-beat and cardiac dulness frequently displaced. When the effusion is on the left side, the semilunar space is usually diminished.

The upper border of the dulness is situated differently, according as the patient lay or walked about during the formation of the effusion. In the first instance, especially among the better classes, the border of dulness runs diagonally from above posteriorly to below anteriorly; if the patient had been walking about (as is frequently the case among hospital patients), the upper border is almost a horizontal line. During the stage of resolution the upper border often forms a convex curve pointing upward, the highest point of which lies in the side of the chest (the curve of *Ellis* and *Damoiseau*). By the change of position of the patient the border of dulness fixed by inflammatory adhesions is altered slowly or not at all.

After a pleuritic effusion is diagnosticated, the nature of the exudate must be determined, whether it is *serous* (simple pleurisy), *purulent* (empyema), or *hæmorrhagic* (usually dependent upon a malignant new growth). Although the patient's condition of strength, the character of the fever, the pulse, and the respiration must be considered in making a differential diagnosis, an (aseptic) *exploratory puncture* with a hypodermic syringe must be undertaken to settle

the diagnosis. It should never be omitted in any case of pleural effusion.

In considering *serous* effusions, it must be noted that it is frequently evoked by a primary disease of the lungs (particularly tuberculosis, next in importance pneumonia, infarct, gangrene, abscess). One should not be satisfied, therefore, with the diagnosis "serous pleurisy," but should examine the lungs, and, if necessary, the sputum, in order to ascertain the etiological factors of the pleurisy. It is important to remember in this connection that many signs of tuberculosis of the lungs lose their importance when found above a pleural effusion; for dulness, bronchial breathing, and crepitant râles may arise as well from compression of the lung.

Aside from diseases of the lung, other things that may produce pleurisy are: diseases of the heart and kidneys, inflammatory conditions of the liver, circumscribed peritonitis, particularly appendicitis, possibly lues; as complications of acute infectious diseases, pleuritis not infrequently arises, just as do inflammations of other serous surfaces (pericarditis, endocarditis).

In *purulent* effusions, the treatment and prognosis are mainly dependent upon the determination of the etiology; aside from the history, many cases require a bacteriological examination of the bacteria contained in the pus obtained from the exploratory puncture.

Pneumococci in the pus denote *meta-pneumonia*, tubercle bacilli *tubercular* empyema. Streptococci and staphylococci give no certain evidence as to the origin of the disease; in tubercular disease of the lungs, empyema dependent upon streptococci and staphylococci may arise. Bacilli of putrefaction are found in empyema in gangrene of the lungs or in embolic infarcts of rotten purulent processes, especially in puerperal infections. *The continued absence of bacteria in the pus speaks for tuberculosis.*

In many cases during and after the resolution of pleural effusions, adhesions between the two layers of the pleura spring up with the formation of thick connective tissue (*pleuritis retrahens*). The *pleuritic thickenings* manifest about the same physical signs as the effusion (dulness, diminution of the respiratory murmur, and the vocal fremitus), yet they differ in this respect, that they show signs

of retraction and of tension on neighboring organs (flattening and drawing in of one side of the thorax, displacement of the heart, or increased size of the semilunar space).

Phthisis pulmonum.—The first stage may give evidence of no physical changes in the thorax. The suspicion of tuberculosis is aroused by various uncertain symptoms: a hacking cough, little expectoration, headache, easy fatigue, loss of appetite, gastric disturbances, loss of flesh and strength; an hereditary taint and the habitus paralyticus make the aspect of the case more serious. The (early) diagnosis can be made with certainty only by the presence of tubercle bacilli in the sputum.

A rise of temperature following an injection of *Koch's* old tuberculin may be counted as one of the signs which enable an early diagnosis to be made. Although convalescents and weakened persons with healthy lungs may also show a rise of temperature after small doses, still the fever induced by the subcutaneous injection of from 1 to 5 mg. of tuberculin speaks very much in favor of tuberculosis. These injections are not without some danger, however, and are scarcely to be commended for use in private practice.

It is important, for diagnostic purposes, to discover possible sources of infection: residence with tubercular patients (wife or husband, brother or sister, other occupants of the same house).

The first appreciable physical signs are: distinct dulness over *one* apex; vesicular respiration with sharpened, prolonged expiration or broncho-vesicular breathing; fine, non-metallic or bronchitic râles.

In a further advanced stage, marked emaciation, profuse cough and expectoration. Sputum large quantity, mucopurulent, often in little balls; contains elastic fibres and tubercle bacilli. Intense dulness over the apex and below the clavicle, bronchial breathing, profuse, coarse râles with something of a metallic ring.

In the final stage, extreme emaciation, very profuse cough and expectoration, sputum in balls and falling to the bottom

of a vessel. Physical examination: in part extensive areas of dulness, in part a loud tympanitic note (even the lower lobe is sometimes attacked), bronchial breathing, tinkling, coarse râles, in places change of sound in the percussion note.

Prognosis in the very beginning with the possibility of thorough treatment, inclined to be good; in the more advanced stages, usually bad. Complication, in the beginning, usually pleurisy; other possible complications: pneumothorax, miliary tuberculosis, tuberculosis of other organs (larynx, intestine, tubercular meningitis, peritonitis, etc.), general amyloid degeneration.

Pneumothorax.—Rarely seen in the healthy (trauma, fracture of ribs, over-exertion); usually secondary to phthisis, gangrene, abscess, perforating empyema, emphysema. The physical signs are:—

Dilatation of the affected side and its immobility in the existing dyspnoëic breathing. Percussion: abnormally loud, deep note (usually not tympanitic), metallic note when the hammer and pleximeter are used. Auscultation: if the cavity-opening is closed, no respiratory murmur; if it is open, amphoric breathing. A fluid effusion usually takes place soon; sero- or pyo-pneumothorax (exploratory puncture); above the fluid, dulness with no respiratory murmur and no vocal fremitus; instantaneous change in the borders of the dulness on change of position. Metallic splashing sound on shaking the thorax from side to side, audible at quite a distance (*succussio Hippocratis*). The prognosis depends upon the primary disease and the possibility of operative interference. Prognosis in the healthy, good.

Fetid bronchitis is diagnosticated when the sputum is foul-smelling (nose and throat not being involved), when no characteristic elements aside from the fetid plugs are formed in the expectoration, and when there is *no dulness* over the lungs and the signs of bronchitis only are present.

The prognosis depends upon the intensity of the bronchitis or upon the presence of bronchiectasis and upon the general symptoms evoked by the putrescence. Fetid bronchitis, with no septic manifestations, usually offers a good prognosis. Fetid bronchitis frequently leads to large bronchiectases which may firmly compress the surrounding tissue. In this manner, a putrid bronchitis may call forth dulness (in the lower lobes).

Gangrene of the lungs is diagnosticated from the putrid expectoration, which contains fragments of lung tissue in addition to fetid plugs (p. 149), and the physical demonstration of the necrotic area of lung: dulness, bronchial breathing, moist râles.

The diagnosis must also establish the *cause* of the gangrene, which modifies the prognosis: trauma (usually great pressure), pneumonia (frequently after influenza), embolus, perforating putrid abscess of the bronchial glands, perforating putrid empyema, extension of gangrenous foci from the œsophagus, vertebræ, intestines, liver.

As far as the prognosis is concerned, there must be determined: (1) the extent of the local disease: in circumscribed gangrene without perceptible formation of cavities, prognosis inclined to be good; in diffuse gangrene with formation of cavities, prognosis bad; (2) the cause of the gangrene: injury and pneumonia, empyema, bronchial abscesses, give comparatively a good prognosis; suppurating emboli, extension from the œsophagus, vertebræ, etc., usually a bad prognosis; (3) the general symptoms: signs of severe infection (very rapid pulse, delirium and collapse) are of evil omen.

Hæmorrhagic infarct of the lung is diagnosticated when in conditions which accompany the possibility of local *thrombosis* (puerperium, marasmus, wounds, decubitus, etc., especially *dilatation of the right heart*) there are sudden stitches in the side, cough, and bloody sputum often accompanied by fever. The diagnosis is made more certain by the demonstration of a circumscribed infiltration (dulness, diminished or bronchial breathing, râles), frequently an additional pleural effusion. The prognosis depends upon the cause of the embolus and the bodily strength; small infarcts, not infected, are readily absorbed.

Abscess of the lung is diagnosticated from purulent sputum containing elastic fibres *without* tubercle bacilli, with remittent fever, when the cause of the abscess (pneumonia, infected infarct, injury) may be simultaneously proven and the signs of infiltration or of a cavity are present. Prognosis dependent upon the cause and the general manifestations; a favorable outcome lies in perforation into a bronchus and subsequent healing.

Tumor of the lung (carcinoma or sarcoma) produces local dullness, over which may usually be heard bronchial breathing or râles. The cutaneous veins in the region of the dullness are usually dilated and frequently the veins of the corresponding arm are involved. In many cases, a sputum like raspberry jelly is expectorated. Sometimes a hæmorrhagic effusion into the pleura occurs, often a swelling of the axillary glands. Cachexia sets in during the course of the disease.

Echinococcus of the lung can be diagnosticated only when the signs of a tumor are present and when echinococcus cysts are found in the sputum.

Syphilis of the lung must be considered from the diagnostic point when a diffuse infiltration or contraction presents itself, when tertiary syphilis is known to be present and tuberculosis is excluded. The proof of the diagnosis lies in the successful issue of specific treatment.

Actinomycosis of the lungs. — Signs of infiltration and cavity formation in the lungs, with considerable secondary pleurisy. The diagnosis is made more positive by the demonstration of the presence of the ray fungus in yellow granulations contained in the sputum.

CHAPTER VII

DIAGNOSIS OF THE DISEASES OF THE CIRCULATORY SYSTEM

For the anamnesis, the following things must be considered: 1. The previous life of the patient: *excessive physical exertion* and great *psychical excitement* cause idiopathic cardiac disease. Too luxurious eating and drinking produce increased blood pressure followed by arterial sclerosis and heart-disease. Too good living produces obesity (*fatty heart*). *Alcoholism* evokes cardiac weakness; too much smoking, neurotic conditions of the heart. 2. Previous diseases: *acute articular rheumatism*, not quite so frequently all the other acute infectious diseases (scarlatina, erysipelas, malaria, etc.), leads to endocarditis or myocarditis. *Syphilis* may be the cause of a myocarditis. 3. Previous symptoms of heart and kidney diseases possibly present.

The diagnosis of diseases of the heart is supported by subjective symptoms: abnormal sensations in the cardiac region, palpitation of the heart, a sense of anxiety; by the presence of dyspnœa, cyanosis, and œdema, and by the results of the physical examination of the heart and blood-vessels.

Complaints of cardiac difficulties (palpitation, sense of anxiety, etc.) without real dyspnœa, without cyanosis and œdema and in the absence of physical anomalies, are to be referred to *nervous* affections of the heart.

Tachycardia, increased rapidity of the pulse, is often seen in nervous affections of the heart in consequence of excitement, excesses, etc., and as a result of certain digestive disturbances and sometimes without appreciable reason (cf. p. 171).

Angina pectoris is a severe pain in the region of the heart com-

ing on in attacks, usually with pain extending toward the left arm and accompanied by the most exquisite sensations of anguish and anxiety. It may appear in any severe cardiac disease, especially in sclerosis of the coronary arteries (*angina pectoris vera seu Heberdenii*). Painful attacks like those of *angina pectoris* may arise in neurasthenics (*angina pectoris neurasthenica*); in these cases there are usually other symptoms on the part of the vasomotor system present, such as alternate flushing and pallor of the face and arms. In every case, *angina pectoris* is to be regarded as a serious matter which demands the closest examination of the circulatory apparatus.

For a discussion of dyspnœa, cyanosis, and œdema, see pp. 10, 125.

Cardiac asthma is the name given to attacks of dyspnœa in cardiac disease, which last for hours, more rarely for days, and which are followed by intervals of freedom; it may appear whenever there is dilatation of the left ventricle. The differential diagnosis as to bronchial asthma rests upon the *establishment of the dilated ventricle* and the small, rapid, irregular pulse.

In attacks of asthma of doubtful etiology the greatest value must be placed on the examination of the heart, particularly the palpation of the apex-beat and the feeling of the pulse. In *bronchial asthma the heart is healthy*. In other respects, the two diseases may simulate each other closely, since in all weakened conditions of the left heart, a bronchitis due to *congestion* and a secondary *emphysema* may arise (expansion of the lung due to distention of the pulmonary vessels). A characteristic difference is often seen in the character of the sputum (see p. 147). In bronchial asthma the expectoration is tenacious, glassy, mucous, with whitish-yellow lumps, containing, on microscopic examination, crystals, spirals, and eosinophile cells; the sputum of cardiac asthma usually shows the presence of blood and approximates in appearance that of œdema of the lungs. Microscopically, alveolar epithelium is often to be seen.

The *œdema* of cardiac disease begins at the ankles and slowly ascends; it attacks last the hands, arms, and face; the œdema of Bright's disease usually begins in the face.

Albuminuria in heart-disease appears only when a high degree of stasis is present; at the same time the urine is scanty and of high specific gravity (see below).

The objective examination of the heart consists of inspection and palpation, percussion and auscultation of the heart and great vessels, examination of the radial pulse and of the urine.

INSPECTION AND PALPATION

The *position and force of the impulse of the heart* and of the *apex-beat* must be determined.

The *impulse of the heart* denotes the impulse of the systole, the elevation of the entire cardiac area; the *apex-beat* is the visible and palpable expansion of the intercostal space furthest to the left, external and inferior.

1. *Position of the apex-beat.* In healthy persons, the hand placed over the cardiac area feels a weak, systolic impulse; the apex-beat is to be felt in the 5th intercostal space midway between the parasternal and mamillary lines.

In children the apex-beat is frequently somewhat higher, in the aged one intercostal space lower; in deep inspiration the apex-beat sinks a little lower. With the patient lying on the left side the apex-beat may be moved a finger's breadth to the left; this is especially true of rapidly emaciating persons, when the patient turns to the right side the apex-beat soon recovers its normal position.

Permanent displacement of the apex-beat is of great diagnostic importance; it denotes either *dilatation* or *compression* of the heart.

Displacement of the apex-beat toward the left signifies (a) *dilatation of the left side of the heart*, (b) *dislodgment of the entire heart toward the left*; in the latter cases a pleural effusion or pneumothorax exists on the right side, in rarer instances a tumor may be demonstrated, or there may be a retracted pleura on the left side.

Displacement of the apex-beat toward the right always depends upon a *dislodgment of the heart*, through a pleuritic effusion or pneumothorax on the left side, or a retracted pleura on the right side.

Displacement of the apex-beat downward may occur through hypertrophy of the left ventricle, less often aneurysm of the aorta, or unusually deep attachments of the diaphragm.

Displacement of the apex-beat upward takes place only when the diaphragm is pushed up in consequence of extraordinary distention of the abdomen (ascites, meteorism, dilatation of the stomach, tumors, pregnancy).

2. *Force of the cardiac impulse and apex-beat.* A diminution in the force of the cardiac impulse and apex-beat to a point at which neither can be felt may occur: (1) in very fat persons; (2) when the lung overlaps the heart: emphysema; (3) when the pericardium is distended with fluid, more rarely when there is a tumor of the pericardium; (4) in all *weakened conditions of the heart*.

In many healthy persons, however, the apex-beat is never felt because it strikes against the rib instead of in the intercostal space.

Increased force of the cardiac impulse and apex-beat. An "elevating" impulse is imparted to the hand. It is present: (1) in *physiologically increased cardiac activity*, when there is psychical excitement, great exertion, in fever; (2) in *hypertrophy of the heart*; (3) often in *dilatation of the heart* when the apex-beat is also displaced outward.

While the diminished force of the apex-beat frequently speaks for cardiac weakness in persons who are not very fat and not emphysematous, its increased force by no means indicates an increased cardiac strength. *Martius* established the fact that the appreciation of the cardiac impulse depends not only upon the functional activity of the heart, but upon the cardiac area exposed to the thoracic wall. According to *Martius*, the systole of the ventricle may be divided into two periods. It first contracts with closed aortic valves; by this manœuvre its form is changed in a typical way, producing the cardiac impulse, but its volume is unchanged (*period of closure*). During the second period of the systole the aortic valves open, the volume of the ventricle is diminished (*period of expulsion*). This is the explanation, too, of the fact that very weak, dilated hearts with a small pulse often

give a decided impulse; during the period of closure a much larger cardiac volume than normally is impelled against the chest-wall, and much less blood is sent into the aorta during the period of expulsion than from a healthy heart.

Sounds or murmurs felt by palpation have the significance as when appreciated by auscultation. The *thrill* palpable over *stenosed* cardiac valves is particularly worthy of note. At the apex a presystolic thrill is characteristic of mitral stenosis; at the right edge of the sternum in the 2d intercostal space a systolic thrill is characteristic of aortic stenosis. (Aneurysm must not be forgotten, however.)

A *bulging of the cardiac area* speaks for dilatation and hypertrophy of the heart or pericardial effusion, although rachitic changes in the bones must be borne in mind.

A *systolic retraction* of the 5th intercostal space by the apex-beat occurs only when the two layers of the pericardial sac are adherent in consequence of chronic pericarditis; the *pulsus paradoxus* is often present (p. 173).

Visible pulsations (synchronous with the radial pulse): over the aorta or pulmonary artery denote aneurysm or an infiltration of the corresponding lobe of the lung; in the *epigastrium*, frequently of no diagnostic significance (when the diaphragm lies low), more often depending upon dilatation of the right ventricle. Visible pulsations of the *liver*, synchronous with a venous pulse, have the same meaning as the actual venous pulse (tricuspid insufficiency).

Venous pulsations, visible at the bulb of the jugular vein or in the jugular vein if the valves of the bulb are insufficient, are either synchronous with the systole of the heart (actual venous pulse, presystolic-systolic), or they precede the cardiac systole (diastolic-presystolic) (cf. p. 176).

An actual venous pulse is the principal sign of tricuspid insufficiency; the presystolic venous pulse is often seen in conditions of venous congestion without valvular insufficiency.

PERCUSSION OF THE HEART

Normal limits of cardiac dulness. The internal border runs along the left edge of the sternum; the external border forms a somewhat convex arch directed outward from the 4th costal cartilage to the 5th intercostal space, between the mamillary and parasternal lines (apex-beat). The upper border lies at the lower edge of the 4th rib, the lower border upon the 6th rib, although this can not always be accurately determined because of the encroaching liver dulness.

The limits described are those of *absolute flatness*, i.e., within these limits the dulness is intense; beyond these limits the so-called "relative dulness" lies, above as high as the upper border of the 3d rib, to the right as far as the median line; but this "relative dulness" is normally not very intense.

In children the area of cardiac dulness is somewhat greater, in the aged somewhat smaller. Every deep inspiration diminishes the area. With the patient lying on the left side, the external border moves outward about one finger's breadth.

The **increased area of cardiac dulness** is one of the principal signs of advanced heart-disease. Lateral extension of the dulness usually denotes dilatation of the ventricle. Dilatation is the *second stage* in most cardiac diseases, and arises from hypertrophy.

1. *Extension of the cardiac dulness to the left* over the mamillary line denotes dilatation of the left ventricle; this is induced by aortic insufficiency or stenosis, by mitral insufficiency, and by the causes of idiopathic heart-disease (see p. 176).

2. *Extension of the cardiac dulness toward the right* beyond the left border of the sternum points to dilatation of the right ventricle, although a similar note evoked over the lower half of the sternum may be due to a mere collection of fat; dilatation of the right ventricle arises in mitral stenosis and insufficiency, in valvular lesions of the right

side of the heart as well as in emphysema, kypho-scoliosis, retracted pleuræ.

3. *Simultaneous expansion of the cardiac dulness toward both sides and upward* denotes a fluid effusion into the pericardium (pericarditis or hydro-pericardium). The area of dulness forms an equilateral triangle the apex of which lies in the 3d to the 1st intercostal spaces.

In every case of enlargement of the area of cardiac dulness, it must be decided whether there is an *actual* dilatation (increase of volume), or whether there is (1) a displacement of the entire heart, (2) a withdrawal of the lung covering the heart so that a greater cardiac area lies directly against the chest-wall. The cardiac dulness is changed in place by pneumothorax, pleuritic effusions, tumors, retracting processes of the pleuræ and lungs; the area is freed from a covering of pulmonary tissue in retraction of the lung; the heart is likewise brought nearer the chest-wall by upward pressure of the diaphragm (ascites, pregnancy, etc.). In cases of transposition of the viscera the entire heart lies on the right side, the liver on the left.

Hypertrophy of the heart is usually *not* demonstrable by percussion; only after dilatation is added to the hypertrophy can one make the percutory demonstration.

Hypertrophy of the left ventricle is diagnosticated by the powerful apex-beat, accompanied by an abnormally high tension of the radial artery, accentuation of the systolic mitral and the diastolic aortic sounds.

Hypertrophy of the right ventricle is diagnosticated by the abnormal accentuation of the diastolic pulmonic sound.

Diminution or absence of the cardiac area of dulness is found when the over-distended lung covers the heart (emphysema).

The entrance of air into the pericardium (*pneumo-pericardium*) produces a tympanitic or metallic percussion note over the area of cardiac dulness; this is a fatal, very rare phenomenon caused by perforation of an ulcer of the stomach or the contents of a lung cavity into the pericardium.

Dulness over the upper part of the sternum, or directly next to it, is diagnostic of aneurysm of the arch of the aorta or of a mediastinal tumor; in very rare cases, of an enlarged thymus gland or of a sub-sternal goitre.

AUSCULTATION OF THE HEART

The auscultation of the heart shows whether or not there are valvular lesions present; these lesions are recognized by characteristic murmurs. Clear heart-sounds denote the intactness of the valves; but despite this fact, the heart may be diseased, hypertrophic, or dilated. *Dilatation and hypertrophy of the heart in the presence of clear tones rest upon disease of the cardiac muscle* (idiopathic heart-disease).

NORMAL AND INTENSIFIED SOUNDS

The sounds of the *mitral valve* are auscultated at the apex, those of the *tricuspid* at the right border of the sternum at the 5th and 6th costal cartilage; the sounds of the aortic valves are best heard at the right border of the sternum in the 2d intercostal space; those of the pulmonic valves at the left border of the sternum in the 2d intercostal space.

Over each valve are heard a systolic sound during the contraction of the ventricles and a diastolic sound during the dilatation of the ventricles.

Over the mitral and tricuspid valves only one sound is heard, the systolic, produced by the tension of the valves and the contraction of the muscles of the ventricles; the diastolic sound is carried away by the aortic in the one case, by the pulmonic in the other. Over the arterial openings, two sounds arise, the systolic, produced by the tension of the expanded vessels, the diastolic, through closure of the valves.

Over the mitral and tricuspid valves, the systolic sound is normally somewhat louder than the diastolic; over the

aorta and pulmonary artery the diastolic is normally somewhat louder than the systolic.

Abnormal *intensification* of the mitral systolic sound in hypertrophy of the left ventricle and in physiologically increased activity of the heart (exertion, excitement); also in fever.

Abnormal *diminution* of the mitral systolic sound in all weakened conditions of the left ventricle (often dilated).

Abnormal *intensification* of the 2d pulmonic sound denotes hypertrophy of the right ventricle.

Abnormal *intensification* of the 2d aortic sound denotes hypertrophy of the left ventricle.

A *musical timbre* in the heart-sounds does not allow of essential diagnostic discrimination; it is usually produced by an increased tension of the flaps of the valves.

A *metallic sound* of the cardiac sounds (often to be heard at some distance) proves the presence of large spaces of air near the heart; also heard in cavities of the lung, dilatation of the stomach, and in the very rare, fatal cases of the entrance of air into the pericardium (pneumo-pericardium).

Reduplication of the heart-sounds is of little diagnostic value. It occurs in the healthy and is particularly frequent in the systolic sounds heard at the apex in hypertrophy following contraction of the kidney. Reduplication of the diastolic sound in consequence of mitral stenosis.

MURMURS

Systolic and diastolic murmurs are distinguished; their nomenclature depends upon the fact whether or not they are synchronous with the cardiac impulse (or pulse) or not. A diastolic murmur which immediately precedes the impulse of the heart is called presystolic. A murmur occurs simultaneously with a heart-sound, or after it, or with no relation to the sounds.

The murmurs are best heard in a direction perpendicular to the current of blood which produces them. Auscultation in mitral insufficiency is therefore practised in the 2d left intercostal space; in aortic insufficiency the diastolic mur-

mur is best auscultated at the middle of the sternum or at the left border of the sternum in the 3d intercostal space.

A *systolic* murmur over the *mitral* denotes insufficiency of the mitral. This murmur may depend upon anatomical changes (endocarditis); it may, however, be functional or *accidental*.

Accidental murmurs are produced by a turning over of the borders of the valve in consequence of undue stretching of the papillary muscles or by relative insufficiency in consequence of dilatation of the ventricle. Accidental murmurs are soft, blowing, usually heard only as *systolic* murmurs.

A *systolic* murmur at the apex is regarded as functional, if the patient has *fever*, is *anæmic* or *poorly nourished*, and the murmurs disappear with time. It may be referred to an endocarditis, when a sufficient etiology can be gathered (especially articular rheumatism) and other symptoms of valvular lesions are present (accentuation of the 2d pulmonary, dilatation of the right ventricle, etc.).

Diastolic (presystolic) murmur over the *mitral* denotes mitral stenosis.

Systolic murmur over the *aorta* is diagnostic of aortic stenosis.

Diastolic murmur over the *aorta* signifies aortic insufficiency caused by endocarditis or arterio-sclerosis.

If *two* murmurs are heard, the greatest importance is to be attached to the diastolic.

Diastolic murmurs are but rarely accidental, while systolic murmurs frequently rest upon a functional basis.

The force and the character of the murmur offer a small basis as to the prognosis of the valvular lesion.

The force of the murmur is only in part dependent upon the severity of the anatomical change; of greater importance are the rapidity of the blood-current, the smoothness or roughness of the walls of the valves. The character of murmurs is described as blowing, grating, scratching, etc.

Pericardial friction sounds are not synchronous with the heart's beat, seem to be nearer to the ear on auscultation than endocardial murmurs, often heard at irregular intermissions (puffing murmurs). They prove the presence of fibrinous deposits on the pericardium (*pericarditis fibrinosa*). They are independent of the respiration, but are influenced by very deep inspiration.

Extra-pericardial friction sounds, arising between the pleura and the external layer of the pericardium, have the character of pleuritic friction sounds, usually of a crackling nature, are dependent for their production upon the respiration, and disappear when the breath is held.

AUSCULTATION OF THE VESSELS

The auscultation of the vessels sometimes helps to establish the diagnosis of a valvular lesion.

The systole of the heart corresponds to the diastole of the vessels; heart's systole = vessel's diastole; heart's diastole = vessel's systole.

The *carotid* is best auscultated at the inner margin of sternocleidomastoid muscle at the level of the thyroid cartilage; the subclavian in the outer portion of the supra-clavicular fossa.

Over the carotid and the subclavian one hears normally two sounds, the first (cardiac systole) arises from tension of the wall of the vessel; the second (cardiac diastole) is transmitted from the aortic valves.

In *aortic insufficiency* a sawing, systolic murmur is heard over the carotids, because of the extraordinarily sudden tension of the walls of the carotid at the instant of the entrance of the current of blood; the second sound is missing, however, as it is not formed by the aortic valves. A systolic murmur may frequently be heard by transmission from the heart in aortic stenosis, mitral insufficiency, and general arterio-sclerosis.

The *more distant* arteries may also be auscultated (the femoral in the groin, the brachial at the bend of the elbow, the radial above the wrist). In healthy persons no sounds or murmurs are heard in these vessels; by pressing the stethoscope upon the artery a murmur produced by pressure may be elicited (arterial diastolic

murmur: by very hard pressure, this is perceived by the ear as a clear sound). An *abnormal sound* is found in aortic insufficiency even in the smaller vessels (in the palm of the hand, in the forearm, etc.). A *double tone* is heard in the *femoral* in aortic insufficiency, mitral stenosis, pregnancy, lead colic.

Actual *murmurs*, audible without pressure, heard over peripheral arteries prove the presence of an aneurysm, and are usually palpable.

Normally, nothing is heard over the veins. The *jugular vein* may be auscultated at the outer margin of the sterno-cleido-mastoid muscle at the level of the thyroid cartilage.

In all cases of anæmia and chlorosis, a loud humming murmur may be heard over the jugular vein, which sounds loudest when the patient turns his head to the opposite side. Over the femoral vein a murmur may be heard only in anæmia of great intensity.

THE PULSE

The frequency of the pulse in healthy adults varies from 60 to 80 beats a minute, in children from 100 to 140, in the aged from 70 to 90.

Slowing of the pulse (*brachycardia*, *pulsus rarus*) is of diagnostic value only under certain circumstances. It is found in the most widely different conditions, evoked by irritation of the vagus, or paralysis of the sympathetic, or irritation or paralysis of the cardiac centres. Brachycardia in exhaustion, after a crisis or at the beginning of convalescence, is especially noteworthy. It is found in *meningitis* (pressure on the brain), in *jaundice* (action of the biliary acids), and in *colic* in which it offers a point in differential diagnosis between it and peritonitis. A slowing of the pulse occurs most frequently in *stenosis* of the aortic and mitral valves among the cardiac diseases, as well as in some idiopathic heart-diseases (coronary sclerosis) and as a result of the action of digitalis.

Rapidity of the pulse (*tachycardia*, *pulsus frequens*) evoked by paralysis of the vagus, irritation of the sympathetic, or affections of the cardiac ganglia. Normal after physical

exertion, psychical irritation, and after eating; pathological in all febrile diseases (with each degree of temperature the pulse is increased 8 beats a minute), common in convalescence from them; in all febrile diseases which lead to consumption (phthisis, anæmia, etc.).

Excessive rapidity (over 160) is a sign of great weakness of the heart (collapse).

Tachycardia is a sign of disturbed compensation in cardiac diseases and is often proportional to the intensity of the disturbance. Tachycardia is also one of the principal symptoms of neurotic disease of the heart, and when it appears in attacks, forms a particular disease (paroxysmal tachycardia).

Tachycardia with exophthalmos, goitre, and tremor of the fingers, frequently associated with general cachexia, forms the symptom-complex of *Basedow's* disease.

The rhythm of the pulse. Irregularity of the pulse (arythmia) is present in many cardiac diseases without offering points for differential diagnosis. Arythmia of mild character is not uncommon among nervous people, after excitement, excesses, gastric disturbances, in constipation; sometimes no reason can be assigned for it. Although arythmia always demands a careful examination of the heart, the diagnosis of a cardiac disease should not be made from this symptom alone.

Embryocardia is the equalization of the systole and diastole through the absence of the normal pause following the diastole. The heart-beats sound like the tick-tack of a clock, like the foetal heart-sounds. Embryocardia is often the expression of a very weak heart.

Characteristic types of irregular pulse are: *Pulsus alternans*: two heart-contractions correspond to one pulse-beat, or the second pulse-beat is but feebly to be felt. *Pulsus bigeminus*: every third beat of the pulse is omitted. *Pulsus trigeminus*: every fourth pulsation is omitted. No diagnostic conclusions can be reached from these irregularities.

Pulsus paradoxus: in deep inspiration, the pulse becomes small or disappears; appears in adhesions of the layers of the pericardium, mediastinitis with cicatrisation or adhesions, mediastinal tumors, stenosis of the air-passages.

Unequal rebound of the pulse at symmetrical arteries, or a *delayed* pulse in corresponding arteries, is a symptom of aneurysm. The former condition may be due, however, to congenital anomalies of the arteries.

Velocity of the pulse (quick or slow): the pulse is rapid or slow according to the rapidity or slowness of the expansion and collapse of the arterial wall. A *rapid pulse* (*pulsus celer*) is found in all conditions of increased cardiac activity, particularly hypertrophy of the left ventricle. It is characteristic of *aortic insufficiency* (*pulsus celer et altus*), contracted kidney, *Basedow's* disease, etc. A *slow pulse* (*pulsus tardus*) is seen in the aged, in aortic and mitral stenosis, in aneurysms.

The **tension** of the pulse (large or small, high or low): the height of the pulse-wave depends upon the strength of the heart, the amount of blood in the arteries, and the tension of the arteries. A high-tension pulse is present in fever, hypertrophy of the heart, especially in aortic insufficiency; a small pulse is a sign of cardiac weakness, characteristic among valvular lesions, of stenosis.

Hardness of the pulse (hard or soft, *durus* or *mollis*): this quality depends upon the *tension* of the arterial walls and is proportional to the strength which must be used by the examining finger to compress the pulse. Hard pulse in hypertrophy of the left ventricle (wiry in contracted kidney), as well as in tetanic contraction of the arterial muscles. Soft pulse in anæmia and fever. Hard pulse in *arterio-sclerosis* due to *deposits of lime* in the walls of the arteries: the artery may be rolled under the finger.

SPHYGMOGRAPHIC TRACINGS (Figs. 30-33)

The purpose of sphygmographic tracings of the pulse-curve is to reach precision in the recognition of the changes in the pulse by *objective* means. A diagnosis may be supported by such tracings.

An ascending and a descending limb is seen in sphygmographic curves. Elevations arising from the ascending limb are called *anacrotic*; arising from the descending limb, *katacrotic*. In the normal pulse of adults, the ascending limb rises almost perpendicularly. Anacrotic elevations appear only in disease of the heart or arteries, the expansion of the arteries occurring in intermissions. The descending limb has normally a decided elevation: the *dicrotic wave*, produced by the falling back of the blood against the aortic valves; and several minor waves (waves of elasticity, tidal and *postdicrotic*) evoked by the elasticity of the arterial wall. The tidal waves are marked when the walls of the artery are under tension, as in lead colic. At the same time the dicrotic wave is small. The tidal wave is very small or disappears altogether in a soft artery under low tension; the dicrotic wave then becomes prominent and is appreciated by the finger as a second, weak pulsation: this is dicrotism of the pulse.

Dicrotism is found in febrile disease, especially in typhoid. In tracings, the dicrotic wave appears in various types according as its record appears above or below the base line: in a *predicrotic* pulse the elevation begins before the descending limb reaches the base line (moderate fever); in a *dicrotic* pulse, as the descending limb reaches the base line; in a *postdicrotic* pulse it begins below the base line (higher fever). In the tracing of a monocrotic pulse (very high fever), no dicrotism is noticeable.

Pulsus tardus shows a slowly rising ascending limb, a rounded summit, no tidal wave, no dicrotic wave (senile pulse). *Pulsus celer et altus* has a perpendicular ascending limb, no dicrotic elevation, several tidal waves.

The venous pulse shows a negative picture of the arterial pulse. The anacrotic limb is long drawn out and possesses a notch (*anadicrotic*), the katacrotic falls almost perpendicularly (*katamonicrotic*). The second limb of the tracing corresponds to the systole of the right auricle, the katamonicrotic limb to the ventricular contraction (auricular diastole). On the other hand, in *insuffi-*



FIG. 30.—SPHYGMOGRAPHIC TRACING OF THE RADIAL ARTERY OF A HEALTHY YOUNG MAN.



FIG. 31.—SPHYGMOGRAPHIC TRACING OF THE RADIAL ARTERY IN AORTIC INSUFFICIENCY.



FIG. 32.—SPHYGMOGRAPHIC TRACING OF THE RADIAL ARTERY IN AORTIC STENOSIS.



FIG. 33.—SPHYGMOGRAPHIC TRACING OF THE RADIAL ARTERY IN MITRAL STENOSIS.

ciency of the tricuspid, the expansion of the vein due to the systole of the auricle does not follow the systolic collapse of the heart, but there is *an additional expansion synchronous with the ventricular systole*. The collapse of the vein takes place in the next diastole. The "actual" venous pulse begins, then, in the ventricular diastole (auricular systole), lasts throughout the entire systole, and ends only at the beginning of the following diastole (cf. p. 164).

THE EXAMINATION OF THE URINE IN CARDIAC CASES

Since the excretion of the urine is dependent, in part, upon the arterial blood-pressure, a diminution of the arterial or increase of the venous blood-pressure is recognized in the *diminished* amount of urine. In cases of cardiac weakness or disturbed compensation the urine is scant, dark-red, of high specific gravity, with profuse brick-red sediment, frequently containing a trace of albumin.

In congestive conditions of long standing, a secondary nephritis may develop with hyaline and granular casts in the sediment; the nephritis of congestion may even lead to granular atrophy.

The improvement in the heart-disease shows itself plainly in the increased amount of urine and the disappearance of the albuminuria.

SYMPTOMS OF THE MOST IMPORTANT CARDIAC DISEASES

Common symptoms to all these diseases are: *in the stage of compensation, absence of any essential difficulties*; in the stage of disturbed compensation: cyanosis, dyspnoea, oedema, urine of congestion.

IDIOPATHIC CARDIAC DISEASES

Hypertrophy, or dilatation of the ventricle, with clear tones, or even the systolic murmur of relative insufficiency.

Prolonged over-activity of the left ventricle in arteriosclerosis, chronic *nephritis* and *contracted kidney, over-exertion*

for a long period, over-indulgence in drink (beer heart),—all these factors aid in production of hypertrophy of the left ventricle with subsequent dilatation.

Cardiac weakness without a previous hypertrophy, partly with and partly without dilatation of the heart (*debilitas cordis*, weak heart), is developed (1) by the action of toxic substances upon the heart-muscle: *alcoholism* (whiskey drinking), excessive use of tobacco, *infectious toxins* (myocarditis after diphtheria, typhoid, etc.); (2) by improper nourishment: inanition, anæmia, senility.

Hypertrophy of the right ventricle, with subsequent dilatation, arises in all obstructions to the pulmonary circulation (emphysema, pleural thickenings, kypho-scoliosis).

A *fatty heart* is the heart which gives disagreeable phenomena to very stout people. Cardiac difficulties of the first degree are those produced by the deposit of fat in the abdomen as well as in the pericardium; difficulties of greater intensity are referable to the growth of fat into the heart-muscle. When the cardiac muscle undergoes fatty degeneration, troubles of the most serious nature are evoked, leading even to death. As fatty degeneration of the heart is mostly the ultimate outcome of fatty deposits about the heart, many writers think that the expression "fatty heart" should be reserved for this final stage, and a term expressive of "cardiac weakness" should be employed for the cardiac troubles of the obese.

VALVULAR LESIONS

Valvular lesions are the result of valvular endocarditis, which is productive of contraction (insufficiency) or adhesions (stenosis) of the flaps of the valves. The endocarditis is usually a sequel of the acute infectious diseases, particularly of articular rheumatism. Besides this etiology, arteriosclerosis may act as a cause.

When the valvular lesion is completed, hypertrophy, and, later, dilatation ensues, in consequence of the forced increase of function on the part of the ventricle. In aortic lesions

it is the left, in mitral lesions the right ventricle which is thus affected.

Aortic insufficiency (frequent). Marked cardiac impulse, apex-beat displaced to the left and downward, cardiac dulness extended to the left. Diastolic murmur in the direction of the blood-current regurgitating from the aorta. Best heard in the middle of the sternum and to the left of it in the 3d intercostal space. Frequently a systolic murmur at the apex and accentuated 2d pulmonic sound. *Pulsus celer et altus*. Strong pulsation of the carotids; systolic murmur in the carotids with absence of the 2d sound. Humming sound in the femoral. Capillary pulse.

Aortic stenosis (rarely alone). Weak cardiac impulse, apex-beat displaced to the left and downward, but much less than in insufficiency. Cardiac dulness extended to the left. Loud systolic murmur over the aorta (frequently *frémissement cataire*); heard much weaker over the other valves. Second aortic sound weak or absent. Pulse small, of low tension, slow. Frequently facial pallor; sometimes syncope, convulsions, attacks of vertigo.

Mitral insufficiency (most common valvular lesion). Cardiac impulse moderately strong, apex-beat frequently displaced to the left. Cardiac dulness extended to the right. At the apex and the pulmonic, a systolic murmur; 2d pulmonic sound much accentuated. Marked dilatation of the right ventricle is accompanied by congestion of the liver and venous pulse.

Mitral stenosis (rarely alone). Cardiac impulse quite marked, epigastric pulsation common. Cardiac dulness extended to the right. At the apex a presystolic murmur (frequently *frémissement cataire*), intensified, rough, systolic sound. Forcible closure of the pulmonic valve. Pulse slow and small, slightly irregular.

Tricuspid insufficiency. Cardiac dulness extended to the right, systolic murmur over the tricuspid valve, diminished

intensity of 2d pulmonic sound, actual venous pulse, pulsation of the liver. The symptoms of this disease are rarely due to anatomical changes in the valve, but are more frequently to be traced to relative insufficiency when marked dilatation of the right ventricle exists as a common sequel of mitral lesions.

The *pulmonic* lesions are always congenital and are quite rare. They are marked by deep cyanosis, the cardiac dulness is extended to the right, and the murmurs heard correspond to the lesion present.

PERICARDITIS

Cardiac dulness increased in area in the form of an equilateral triangle, the apex of which points upward. The upper limit of cardiac dulness, 2d to 3d rib. Cardiac impulse and apex-beat weak or not palpable; increased in intensity when the patient bends forward, and are usually felt somewhat internal to the area of dulness. Heart-sounds very weak. Friction sounds not synchronous with the cardiac beat are common. Pain in the region of the heart not uncommon in swallowing.

Pericardial plates (*concretio pericardii*), after the healing of pericarditis, sometimes lead to the symptoms of cardiac weakness without offering percutory or auscultatory signs. In some cases the diagnosis is made possible by the systolic retraction of the region of the apex-beat, or by the presence of pulsus paradoxus.

SYMPTOMS OF THE PRINCIPAL DISEASES OF THE LARGE BLOOD-VESSELS

Arterio-sclerosis (atheroma of the arteries). — The palpable arteries (especially the radial and temporal) hard and tortuous, sometimes uneven and rough. Pulse under tension, mostly sluggish. Hypertrophy and dilatation of the left ventricle. Frequently a systolic murmur at the apex; less

often a diastolic murmur over the aorta. Angina pectoris or cardiac asthma, common (coronary sclerosis).

Atheroma of the peripheral arteries does not always indicate atheroma of the large vessels and of the heart. On the other hand, a high degree of sclerosis of the aorta (endaortitis) may be present, without any apparent disease of the radial arteries.

Aneurysm of the thoracic aorta. — Dulness over the upper part of the sternum and its neighborhood. Over this area a thrilling systolic or diastolic murmur, audible and palpable. As the disease advances, the pulsating tumor appears to the left of the sternum in the region of the 2d or 3d rib. Frequently hypertrophy and dilatation of the left ventricle. Unevenness of the two radial pulses.

CHAPTER VIII

THE EXAMINATION OF THE URINE

By the examination of the urine we obtain information of :—

1. *The condition of the kidneys* and of the bladder. The normal renal epithelium does not permit the albumin in the blood to pass. In kidney disease, albumin and histological elements appear in the urine. Diseases of the bladder are associated with definite processes of urinary decomposition.

2. *The progress of the metabolic phenomena.* The end products of the disintegration of the albuminoids (urea, etc.) are voided in the urine; from an examination of this excretion we can determine the quantitative relations existing between the nitrogenous income and expenditure, which undergo specific changes in disorders of metabolism, as well as the admixture of certain definite compounds which result from certain anomalies of the metabolic processes, or which escape the chemical change or conversion which they should normally undergo (sugar, acetone, etc.).

3. *The force of the heart* (see p. 176).

4. *Diseases in other organs*, which permit certain substances to escape into the blood and from it into the urine. In disease of the liver, biliary coloring matter, in disease of the intestine, indican, and in suppuration, peptone, appear in the urine.

5. *The presence of heterogeneous compounds* which may be administered from without, such as iodine and mercury.

The quantity of urine passed in 24 hours amounts on the average to 1500 cc., and depends within further limits on the amount of fluids partaken of. A daily quantity below 500 cc. and above 3000 cc. is usually an indication of disease.

Diminution of the amount of urine occurs when there is profuse perspiration and in cases of diarrhoea, in fever, in cardiac weakness, in acute and often in chronic nephritis, in cases of effusions and transudations.

In cardiac diseases and in acute nephritis the prognosis as to immediate danger is for the most part made from the amount of urine passed. In effusions, *e.g.*, pleuritic, the first indication of the absorption of the fluid is shown by an increase of the secretion of urine. The excretion of a small amount of urine is often a sign of *deficient nutrition*.

Increase of the amount of urine is observed in diabetes mellitus and diabetes insipidus, in atrophic (small) kidney, in cases of effusions and transudations when absorption occurs, and often during the period of convalescence from an acute disease.

The **specific gravity** of the urine varies in health between 1010 and 1025, and is in inverse proportion to the amount excreted.

An unusually low specific gravity occurs in contracted kidney and in diabetes insipidus. - A very high specific gravity is observed in diabetes mellitus, in fever where the amount passed is small, in wasting diseases, and in nephritis.

It is possible to determine from the specific gravity the amount of the solid constituents in 1000 cc. of the urine in this manner: multiply the last two ciphers of the specific gravity by 2.33 (*Häser's* coefficient). For example, should the urine to be examined have a specific gravity of 1015, then $15 \times 2.33 = 34.95$; hence the solid constituents in the urine are 34.95 in every 1000 cc.

The color of the urine. — The normal color of the urine is a more or less deep yellow; the smaller the quantity the deeper the color. It is of a *deep yellowish-red* color when the urine contains urobilin (see p. 192), and it is *red* (like *beef solution*) when it contains *blood* (see p. 190). A *brown* color with a yellow foam indicates the presence of *biliary coloring matter* (see p. 191). In *carbolic acid*, seldom in *salicylic acid*, *intoxication* the color varies from an *olive-green* to a *black*. It is *yellowish-green* after the use of *rhubarb* and *santonin*. The color grows darker when exposed to the air, either because melanin is present or because carbolic acid was taken (see p. 202).

The color directs our attention to the abnormal elements which the urine contains, whereupon these may be detected by a chemical and microscopical examination.

Cloudy urine. — Normal urine is clear. The significance of cloudy urine depends on its *reaction*, which may be tested by litmus and by the smell (see below).

Cloudiness of *acid* urine is owing either to the urates which may be present, and in this case the urine becomes clear on heating in a test-tube, or to *organic histological elements* (see below) when the urine will not become clear on heating. These anatomical elements may be detected by the use of the microscope.

The organic elements consist of casts, renal epithelium, or pus cells. Pus is detected by boiling with potassic hydrate, which produces a mucilaginous mixture.

Cloudiness of *alkaline* urine arises from the presence of phosphates, seldom from oxalate of lime, or from organic elements which may be determined by the use of the microscope.

On the addition of an acid the cloudiness arising from the presence of salts will disappear, whereas it will naturally not be dispelled when organic elements give rise to it. Pus is determined here also by the mucilaginous appearance of the urine when boiled with potassic hydrate.

The reaction. — Normal urine is acid. Should the urine to be examined be alkaline, we must determine whether this reaction be due to the presence of a fixed alkali (calcic carbonate) or to a volatile one (ammonium carbonate). This is determined by holding over the urine a piece of moistened red litmus paper. Should this paper turn blue without agitating the urine, then the alkalinity is due to the ammonium carbonate in the urine; should, on the other hand, the blue color result only after dipping the paper into the urine, the alkalinity is due to the presence of *potassium* carbonate or

sodium carbonate. If we hold a glass rod moistened with hydrochloric acid over a dish containing ammoniacal urine, a white cloud of ammonium chloride, NH_4Cl (sal ammoniac), will be produced.

The reaction of the urine depends upon the quantitative relations existing between the acids and bases. Urine contains the following acids: hydrochloric, sulphuric, phosphoric, uric, kreatin, a little hippuric and oxalic; the following bases are present: potassium, sodium, calcium, magnesium, ammonium, kreatinin, xanthin, and hypoxanthin.

By the disintegration (metabolism) of the albuminoids, of the lecithins, and of nuclein, sulphuric acid and phosphoric acid are copiously produced; it is on this account that after the ingestion of meat, cheese, legumens, and cereals, the urine becomes strongly acid.

While *hydrochloric acid* is forming compounds in the stomach, in the first act of the digestion of a meal rich in albuminoids the acid reaction of the urine is diminished; should the hydrochloric acid be removed by vomiting or by washing out the stomach, the urine must likewise be less acid and more alkaline.

A direct increase of alkalinity occurs in the administration of those *potassium salts whose acids are easily oxidized*, potassium tartrate, citrate, and malate, which are oxidized into potassium carbonate. These salts are abundantly present in fruits, berries, and in potatoes. The eating of large quantities of fruit will, therefore, render the urine alkaline, as will naturally the medicinal administration of sodium carbonate.

The alkaline reaction of the urine from presence of a fixed alkali appears soon after a hearty meal; after a hearty indulgence in fruits, berries, or potatoes; in diseases of the stomach where vomiting occurs or where the stomach has been washed out; in cases of absorption of alkaline effusions; after the use of alkaline waters and medicines.

The alkaline reaction of the urine from the presence of a volatile alkali is produced by the development of bacteria in the urine, which decompose the urea into ammonium carbonate, thus $(\text{CONH}_2)_2 + 2\text{H}_2\text{O} = \text{CO}(\text{NH}_4)_2$. This decomposition takes place in all urine that has stood for a long

time, especially in a warm place. Should the urine be decomposed as soon as it is voided from the bladder, there is a *cystitis* present.

Ammoniacal decomposition produces a very characteristic odor, which is easily recognized.

Every alkaline urine contains a sediment composed of ammonio-magnesium phosphates (triple phosphates), calcium phosphate, and often of calcium carbonate. In decomposition the triple phosphates are most prominent, and at the same time the urine is rich in bacteria. When the alkalinity is due to a fixed alkali, the sediment contains much calcium phosphate and calcium carbonate.

THE CHEMICAL EXAMINATION OF THE PATHOLOGICAL ELEMENTS OF THE URINE

Albumin. — The presence of albumin (serum albumin and serum globulin) in the urine indicates a lesion of the renal epithelium. Under this head it may deal with mild or intense disturbances of nutrition (a congestion, an anæmia, cloudy swelling, fatty metamorphosis). Pronounced persistent albuminuria indicates nephritis.

In all urine of human beings nominal traces of albumin may be found, still these can only be detected by especially fine methods of analysis (*normal albuminuria*). Small quantities of albumin appear in some persons as a *transient* constituent of the urine after a meal rich in albuminoids, after intense physical strains, after hot baths, and after psychical excitement. It is then known as *physiological albuminuria*.

If the urine contain *blood* or *pus*, the albumin derived from their plasma is dissolved by the urine and even after filtration the urine will show the reaction for albumin; such an albuminuria does not denote in itself a disease of the kidneys. It is known as *spurious albuminuria*.

The examination of a patient is incomplete without an examination of the urine for albumin. When a small quan-

tity is detected, the urine should be again examined from specimens passed at different periods of the day (the urine passed on awakening in the morning is often free from albumin even when the kidneys are diseased).

Of practical significance is *intermittent albuminuria*, which is the condition obtaining when small quantities of albumin appear *transiently* in the urine at different times and mostly in otherwise healthy young persons. Even if the suspicion of nephritis must be entertained, the progress of the disorder is most favorable. *Cyclical albuminuria* occupies a peculiar position; it consists in this: the urine in the early morning is always free of albumin, but shows, if examined every two hours, varying amounts of albumin, which reach the maximum mostly at night. Though in these cases the question of a chronic nephritis can not be excluded with absolute certainty, still a more frequent disappearance of albumin is observed.

TESTS FOR ALBUMIN

Freshly voided urine should be used and preferably the last portion of the urine which has been passed.

For the preparation of the tests the urine should be *clear*, it should be *filtered*.

1. *The boiling test.* The urine is placed in a test-tube to one-quarter of its length and is then heated in the flame until it boils, when $\frac{1}{5}$ volume of nitric acid is added. Should the urine have become cloudy and the cloud have become cleared up during the heating, the cloudiness was due to the presence of acid sodium urate. Should the cloudiness exist on heating and become dispelled only after the addition of nitric acid, it was due to the presence of either calcium carbonate or calcium phosphate. Persistence of the cloudiness or the appearance of a precipitate after the addition of the nitric acid indicates albumin.

Should the portion tested in this manner be permitted to stand in the test-tube over night, the precipitate will have gravitated to the bottom of the tube. The volume of the precipitate in its relation to the volume of the original fluid tested will give an approximate quantitative test. This relation is as follows: slight cloudiness of the fluid (trace of albumin) corresponds to 0.01 per cent.; when the precipitate occupies merely the bottom of the tube, there is 0.05 per cent. of albumin; when the precipitate is

$\frac{1}{10}$ of the volume of the tube contents, there is 0.1 per cent. of albumin; when $\frac{1}{2}$ of the volume, 0.25 per cent.; $\frac{1}{3}$ of the volume, 0.5 per cent.; $\frac{1}{4}$ of the volume, 1 per cent.; when the whole contents become solid, between 2 and 3 per cent. are present.

2. *Test with acetic acid and potassium ferrocyanide.* To the cold urine in a test-tube a few drops of acetic acid,¹ then some of a 5 per cent. solution of potassium ferrocyanide is added drop by drop; if albumin be present there will be immediately or in a few moments a flaky precipitation of the coagulated albumin.

3. *Heller's test.* Concentrated nitric acid is slowly added to a small quantity of urine contained in a test-tube. The tube should be held inclined that the acid may flow down the wall of the tube. The acid will sink to the bottom and at the dividing line between acid and urine, if albumin be present, a narrow cloudy white ring will be formed. Still a similar ring may be produced in the presence of uric acid, urea nitrate, and of the resinous acids which result from the administration of resinous substances like turpentine, copaiba, etc. The ring formed from the presence of the resinous acids is soluble in alcohol, that from urea forms only after long standing and is also distinctly crystalline; the uric acid ring is not as sharply defined as is the ring arising from the presence of albumin, it rises higher in the urine and is only produced in very concentrated urine.

Quantitative determination of the albumin. For clinical purposes a sufficiently accurate quantitative analysis may be made by means of *Esbach's* albuminometer. The albuminometer is filled to the line marked *U* with the urine to be examined, the *Esbach* fluid, which consists of citric acid 5, picronic acid 2.5, distilled water 245, is then added to the line marked *R*, the mixture is then shaken, and on the following day the deposit which has collected will reach a line at the lower portion of the tube, which is graduated, denoting in grammes the amount of albumin present in 1000 cc. of urine.

For scientific purposes, *weighing* of the precipitated albumin is the best method. This method consists of placing 100 cc. of urine in a porcelain dish, adding sufficient acetic acid to render the urine slightly acid, and then heating the contents of the dish to the boiling-point. The fluid containing the precipitate is then filtered, the filter having previously been thoroughly dried and weighed. The precipitate is well washed with hot water, then with alcohol

¹ Sometimes a cloudiness develops on the addition of the acetic acid; this is due to mucin or nucleo-albumin, and should be removed by filtration.

and ether; it is dried and weighed; the additional weight will represent the percentage amount of albumin.

In addition to the albumin precipitated by boiling, the urine may contain forms of albumin which are *not precipitated by heat*. These forms also result in gastric digestion of albumin. It has recently been proven that the end product of the gastric digestion of albumin (*Kühne's peptone*) does not appear in the urine, but that only premature stages of the same (albumoses and propeptones) are there found. It is a mistake, therefore, to speak of a *peptonuria*; we ought to call that condition *albumosuria*. However, this term has up to this time been very little used.

The albumoses (peptone in the old sense) are not precipitated by heat and by acids; their presence is determined by the biuret test after the other albuminous bodies have been precipitated and removed by filtration.

The albumoses are found in urine, especially in cases of absorption of *purulent* or fibrinous exudations (*pyogenic albumosuria*), especially in cases of pneumonia shortly before and after the crisis, in purulent meningitis, in peritonitis, in empyema, at times in cases of intestinal ulceration, in many diseases of the liver, in puerperal fever (*enterogenous*, *hepatogenous*, and *puerperal albumosuria*). Albumosuria is chiefly diagnostic of suppuration, but should be estimated only with great precaution.

Test for peptone (Hofmeister's test). Add to 500 cc. of urine 50 cc. of a concentrated solution of sodium acetate; drop slowly into this a concentrated solution of ferric chloride, until the resulting fluid remains at a red color. At this stage carefully add a solution of potassium hydrate, until the previously strongly acid mixture becomes neutral, or is only slightly acid; then boil and filter after the fluid becomes cool. Should the filtered solution be found by the acetic acid and potassium ferrocyanide test to be free from albumin, the *biuret test* is then used; a few drops of potassium hydrate and a few of a 1 per cent. solution of cupric sulphate being added, a beautiful red color results if peptone be present.

The recently proposed method of *Salkowski* is much simpler and just as sensitive: 20 to 50 cc. of urine which has been freed from albumin, and to which some hydrochloric acid has been added, is precipitated by phospho-molybdic acid; the precipitate is heated, washed with water, finally dissolved in a weak solution of sodium hydrate, and again heated until it assumes a yellow color. After cooling, the biuret test is put into use (see above).

Blood. — We recognize the presence of blood by the color of the urine. This is bright red with a greenish shimmer (like meat solution) in the presence of *oxyhæmoglobin*; dirty brownish-red in the presence of *methæmoglobin*. Nevertheless, we ought not decide that blood is present from the color alone; a *microscopical* examination of the sediment (see p. 210) and the chemical tests for blood should also be made. A spectroscopic examination is only seldom used (Chap. XI.).

Heller's test. To the urine in a test-tube $\frac{1}{4}$ of its volume of potassium hydrate solution is added, and the mixture is boiled. After a short time the earthy phosphates precipitate (magnesium and calcium phosphates). When blood is present the flaky precipitates will be of a reddish-brown color (in normal urine they are grayish-white). The color is best determined after the precipitate has fully collected together.

Van Deen's test (guaiac test). Add to the urine 2 cc. of tincture of guaiac and 2 cc. of old oil of turpentine, then shake the mixture thoroughly; if blood be present, the whole solution will become blue in a short time (pus will give the same reaction). Instead of old turpentine oil the following mixture may be used (*Hühnerfeld's test*): glacial acetic acid, 2; distilled water, 2; oil of turpentine, 100; absolute alcohol, 100; chloroform, 100.

Blood in the urine signifies a *hæmaturia* or a *hæmoglobinuria*. *Hæmaturia* indicates the presence of the *red blood corpuscles* in the urine; the blood may come from the kidney,

the pelvis of the kidney, the bladder, or the urethra. The causes of hæmaturia are acute nephritis, renal calculi, hæmorrhagic infarct of the kidneys, tumors of the kidneys, renal hæmophilia, pyelonephritis, acute cystitis, carcinoma of the bladder, and stone in the bladder.

Temporary hæmaturia may also be the result of physical over-exertion.

In making a differential diagnosis the following data should be considered: the urine is small in amount in acute nephritis and contains more albumin than the amount of blood it contains would justify; in addition to these facts there are casts in the deposits, and the patient, as a rule, has dropsy. Hæmorrhagic renal infarction is associated with cardiac disease, and is accompanied by fever and pains, the blood disappearing from the urine in a few days. The hæmorrhage from renal calculi is preceded by attacks of colic, and the calculus may be passed with the urine. In pyelonephritis a purulent deposit will also be present in the urine. In cystitis there is pain in the bladder and vesicular tenesmus, while the urine contains pus at the same time. In cases of tumor it is sometimes possible to find small particles of the growth in the urine.

When a careful consideration of all the causes mentioned fails to discover the origin of the hæmaturia, it may be possible that, as occurs in rare instances, the hæmorrhage arises from a healthy kidney, of which two varieties are known.

1. The *hæmaturia of bleeders* (renal hæmophilia) may be diagnosticated when the patient's family is one of bleeders, and when he himself has given previous evidence of hæmophilic dyscrasia. 2. *Angioneurotic hæmaturia*, which occurs in neuropathic individuals; in which case one may succeed in stopping the hæmorrhage by suggestion or hydragogue measures. Hæmorrhage from a normal kidney should only be considered when every other diagnostic possibility has been excluded.

In doubtful cases, severe pain in the lumbar region may indicate the origin of blood as coming from the *kidney*; proof of this is the presence of red blood corpuscles in the urinary sediment, when they occur as blood casts.

We can occasionally determine whether the hæmorrhage comes from the bladder by catheterizing and washing out the bladder after micturition has occurred. If the blood comes from the bladder, the returning water will be blood-stained. In spite of this aid it is at times extremely difficult to determine whether the source of the blood is from the kidney or bladder. In such cases the source of the bleeding may be determined by the *cystoscope* in expert hands. The cystoscope will also determine from which kidney the blood arises.

Hæmoglobinuria is the appearance of a solution of the coloring matter (*without* the corpuscles) of the blood in the urine. It results in consequence of the red blood corpuscles being dissolved from various causes, namely, from poisons (potassium chlorate, mussels poisoning, etc.); it appears after transfusion of blood, it results from burns and as a specific disease (periodical hæmoglobinuria), and often after exposure.

Biliary coloring matter appears in the urine either reduced as *hydrobilirubin*, the same as *urobilin*, or unreduced in the form of *bilirubin*. The special biliary coloring matter is detected by the beer-brown color of the urine and the yellow color of the foam which is produced by shaking. Urine containing urobilin is yellowish-red with a tinge of orange. For a positive determination special chemical and spectroscopic analyses are required.

Gmelin's test for bilirubin. Concentrated hydrochloric acid is mixed in a test-tube with one or two drops of fuming nitric acid. The urine is then carefully poured down the walls of the tube; the acid and the urine form two distinct layers, and at their junction a colored ring will be formed; this ring is first green (*biliverdin*), then violet, then red, then yellow (*choletelin*), and finally it assumes a dirty color. The production of a blue ring arises from the presence of *indican*.

The same diagnostic conclusions should be formed when bilirubin is shown to be present in the urine as would from the presence of icterus.

Test for urobilin. Render the urine alkaline by adding some ammonia to the urine in a test-tube, then add 8-10 drops of a 10 per cent. solution of zinc chlorate and filter quickly. View the filtered solution against a dark background, when it will appear green, but by transmitted light it will be rose-red. *Spectroscopical* examination of the urine will detect the smallest portion by showing an absorption line between the green and blue fields.

Hydrobilirubin (urobilin) occurs in many cases of icterus in which the color of the skin of the patient is a dirty yellow (urobilin icterus), in states of passive congestion, and in high fever. In addition it occurs where large extravasations of blood have been absorbed (as a result of the reduction of the coloring matter of the blood identical with bilirubin). With a sufficient regard for the other causes a diagnosis of internal hæmorrhage (apoplexy, infarct, etc.) may be justified from the presence of a large amount of urobilin in the urine.

Sugar.—Urine which contains sugar is usually passed in large amounts; it is of light color, and of a high specific gravity.

The proof of sugar in the urine depends upon its following characters:—

1. Glucose is colored brown (caramel formation) when boiled with potassium hydrate.
2. Glucose has the property to reduce other bodies under high temperatures.
3. Glucose ferments in the presence of yeast and is converted into alcohol and carbonic acid ($C_6H_{12}O_6 = 2C_2H_5OH + 2CO_2$).
4. Glucose turns the plane of polarized light to the right.
5. Glucose unites with phenyl hydrazin into a characteristic chemical compound (glucosazon).

QUALITATIVE ANALYSIS FOR SUGAR

1. *Moore's test* consists in adding to the urine in a test-tube $\frac{1}{2}$ of its volume of potassium hydrate solution and boiling the mixture several times in succession; in the presence of sugar (glucose) the urine will assume a *brown color*.

2. *Reduction tests.* a. *Trommer's test* consists in adding to the urine $\frac{1}{2}$ of its volume of potassium hydrate solution and again adding enough of a 10 per cent. solution of cupric sulphate until the blue precipitate which is formed thereby is entirely dissolved while agitating the mixture; as soon as the addition of another drop of the copper solution is precipitated and is not dissolved by agitating the contents of the tube, stop the addition of the cupric sulphate and heat carefully the upper part of the solution in the tube by holding that part of the tube over a flame. If sugar be present, a yellowish-red precipitate will be formed as soon as that part of the mixture rises to 60° to 70° C. before the boiling-point is reached. As soon as the precipitate forms, stop the heating.

The reaction proceeds as follows: $\text{CuSO}_4 + 2 \text{KHO} = \text{Cu}(\text{HO})_2$ (cupric hydroxide) + K_2SO_4 . Cupric hydroxide, $\text{Cu}(\text{HO})_2$, in itself may be converted by heat into black cupric oxide, $(\text{CuO}) + \text{H}_2\text{O}$. The 2CuO , or the $\text{Cu}(\text{OH})_2$, gives up by the heat one atom of O to the sugar, thus producing Cu_2O , yellow copper oxydul (cupro-oxyd), or CuOH , brown copper oxydulhydrate (cuprohydroxyd) respectively.

The ordinary dissolving of the cupric hydroxide with the production of an *azure-blue* color does not determine the presence of sugar; for the same reaction may be produced when albumin, ammonia, and other organic substances are present.

Even the change of color of the tested solution to yellow *without the precipitation* of a yellow precipitate does not prove the existence of sugar; for uric acid and kreatinin will likewise reduce the oxide of copper, but retain copper oxydul (cupro-oxyd) in solution.

In the strict acceptance the production of copper oxydul does not prove the existence of sugar, but *only that a reducing substance is present in the urine*. Other reducing substances may occur in the urine especially after the ingestion of certain substances (chloral hydrate, camphor, chloroform, turpentine, benzoic acid, salicylic acid, copaiba and cubebs). When any of these have been taken, in addition to *Trommer's test*, a control analysis for sugar should be made by fermentation and the polariscope.

Trommer's test may be made more quickly by adding slowly an

equal volume of a doubly diluted *Fehling's* solution to a few cubic centimetres of the boiling urine. If sugar be present, a yellow precipitate will be thrown down. The *Fehling's* solution, however, should be previously tested to see if it itself will not form a similar precipitate on boiling.

b. Böttcher's test. Saturate 10 cc. of urine with some finely powdered sodium carbonate, add thereto a very small quantity of *basic bismuth nitrate*, and boil for some minutes. The production of a *black color* in the urine indicates the presence of sugar; only when organic substances (albumin, mucus, pus, and blood) are present in the urine may this production of bismuth sulphide lead to error. A very convenient method for conducting this test is by the use of *Nylander's solution*, which consists of Rochelle salts (potassium sodium nitrate, 4; 10 per cent. solution of sodium hydrate, 100; bismuth subnitrate, 2; heated together and then filtered). If urine to which $\frac{1}{10}$ of its volume of *Nylander's* solution has been added be boiled, should sugar be present a brown or black color will be produced.

c. Rubner's test. Add to the urine a knife tip full of plumbic acetate; a precipitate of plumbic phosphate and plumbic sulphate will be formed. Remove the precipitate by filtering and to the filtered solution add some ammonia, whereupon a white precipitate of plumbic oxide will be formed. On boiling at this point of the procedure the white precipitate will become rose-red in the presence of sugar, owing to the formation of a higher oxide (red oxide of lead, Pb_2O_4).

3. *Fermentation test.* This test is one of the most reliable of all tests for sugar. It is performed as follows: shake the test-tube containing the urine to which a small cube of fresh compressed yeast has been added, then pour this mixture into a *fermentation tube*; turn the tube so that the long arm will be filled completely by the urine. When the apparatus has been filled, pour some mercury into it in order to close the long arm, then place the entire apparatus in a warm place at about 24°C . Should the urine contain sugar, gas bubbles composed of CO_2 will arise in the tube in a few hours. That this gas is truly CO_2 may be proven by the addition of potassium hydrate, which will quickly absorb the gas, For a control of this test, two more fermentation tubes should be prepared, one with a mixture of a solution of glucose and yeast (to determine that the yeast is efficient), the other with normal urine and yeast (this should not generate gas, and should show that the yeast itself is free from sugar).

A fermentation tube may be improvised from a test-tube, as may be seen from Fig. 34. The picture shows a fermentation tube made after *Moritz's* suggestion and needs no further explanation. *Pavy's* fermentation tube may be prepared by introducing a glass tube through the stopper of a test-tube which has been completely filled with the urine to be examined; the tube should reach almost to the bottom of the test-tube. The outer end of the tube should be bent and placed in a vessel containing water; the gas which forms collects under the stopper, and by pressure the urine rises in the tube to escape into the vessel containing the water.

4. *Phenyl hydrazin test* is carried out as follows: to 10 cc. of a heated 10 per cent. solution of sodium acetate, as much acid phenyl hydrazin is added as will go on the end of a knife; to this mixture 10 cc. of urine are added, and the test-tube containing the entire solution is placed in a water bath at 100° C. for an hour. When sugar is present, numerous yellow crystals of phenyl glucosazon are formed and precipitated. The latter compound melts at a temperature of 205° C. Should the urine contain but little sugar, the solution is centrifuged, and the sediment examined microscopically for the crystals.

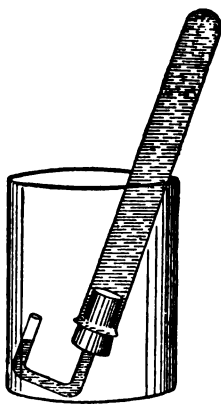


FIG. 34. — IMPROVISED FERMENTATION APPARATUS. (MORITZ.)

Sugar may appear in the urine in health after the ingestion of any meal containing more than 150 g. of glucose (alimentary or physiological glycosuria). In general a suspicion of a diabetic tendency should be held in every case of glycosuria, even when sugar in the urine is transient and only small in amount.

Alimentary glycosuria has sometimes been observed after the ingestion of moderate amounts of glucose (about 100 g.) in the obese, in drunkards, in cases of *Basedow's* disease, as well as in traumatic neurosis, and also in those who have used thyroid preparations. In excessive diuresis a small quantity of sugar may appear in the urine.

The persistent or long-continued appearance of more than $\frac{1}{2}$ per cent. of sugar in the urine occurs only in *diabetes mellitus*. A complete diagnosis of this disease demands the quantitative determination of the amount of sugar passed (see Chap. X., Diseases of Metabolism).

QUANTITATIVE ANALYSIS OF SUGAR IN THE URINE

Estimation of the amount of sugar by Moore's test. Urine heated with potassium hydrate is of a straw-yellow color when it contains about 1 per cent. of sugar, amber-yellow with 2 per cent., brown, like Jamaica rum, with 5 per cent., and dark brown with 7 per cent. This estimation is but little reliable, and will give to the one who is expert in its use only approximate results.

Estimation of the amount with Fehling's solution. *Fehling's* solution consists of 34.639 g. of crystallized cupric sulphate, 173 of Rochelle salts, 100 c.c. of officinal sodium hydrate solution, and water to make the entire mixture 1000 c.c.; 1 c.c. of this solution is reduced by 5 mg. of glucose.

Two cubic centimetres of *Fehling's* solution are put into a test-tube containing 20 c.c. of water, by means of a pipette. The cupric oxide contained in this amount is reduced by exactly 1 cg. of sugar. Bring the contents of the test-tube to a boil; then add *drop by drop* the urine to be examined, and see by transmitted light after every drop whether the solution has become *decolorized*. As soon as it has, stop adding the urine. The number of drops of urine which have been added to produce decolorization will then have contained 0.01 g. of sugar. Twenty drops are regarded as equal to 1 c.c. of urine.

The following table gives the approximate relation of drops to percentage of sugar.

DROPS	PERCENTAGE	DROPS	PERCENTAGE
100	0.20	14	1.40
90	0.21	13	1.50
80	0.25	12	1.60
70	0.28	11	1.80
60	0.30	10	2.00
50	0.40	9	2.20
40	0.50	8	2.50
30 } 25 }	0.60	7	2.80
20	1.00	6	3.30
19	1.05	5	4.00
18	1.10	4	5.00
17	1.15	3	6.60
16	1.20	2	10.00
15	1.30	1	20.00

The results of these values are pretty reliable when the method is properly used.

Titration with the use of Fehling's solution. Strongly dilute 20 c.c. of *Fehling's* solution in a porcelain dish; dilute the urine ten times and place it in a graduated burette. Drop the urine slowly into the boiling *Fehling's* solution until all the copper has been precipitated and the solution has become entirely decolorized. The amount of sugar in the urine necessary to perform this reduction is 0.1 g.; hence the amount of urine used contains 0.1 g. of sugar. From this the percentage of sugar is easily reckoned. For instance, if 27 c.c. of the ten times diluted urine were used to reduce 20 c.c. of *Fehling's* solution, then 0.1 g. of sugar was contained in the 27 c.c. of the ten times diluted urine; therefore in 100 c.c. of the same dilution of urine there would be $\frac{0.1 \times 100}{27} = 0.37$; but since the urine was diluted ten times,

the total percentage would be $10 \times 0.37 = 3.7$ per cent. In order to accurately judge the time to stop adding the urine, a small amount of the *Fehling's* solution that has been used is filtered and added to some potassium ferrocyanide solu-

tion; should some unreduced copper be still held in the solution, a brown color (cupric ferrocyanide) will be produced. The titration method is entirely accurate, but may lead to error on account of the other reducing substances (see p. 193) contained in the urine.

Estimation by Einhorn's saccharimeter. This instrument is the ordinary fermentation tube, whose long arm is empirically graduated. 10 c.c. of urine are mixed in a test-tube with a piece of compressed yeast about the size of a bean; the mixture is well shaken and poured into the fermentation tube. According to its specific gravity, the urine should be previously diluted; a specific gravity of 1018 to 1022 necessitates a double dilution, 1022 to 1028 a fivefold dilution, 1028 to 1038 a tenfold dilution. This method gives a pretty good result for urine containing less than 1 per cent. of sugar, but it is not always accurate in estimating the percentage of sugar in urine containing more than 1 per cent. However, it is quite serviceable for practical purposes, especially for continued estimations.

Determination of the specific gravity before and after fermentation. The temperature and the specific gravity of the urine are taken; then 100 to 200 c.c. of urine are placed in a flask containing some compressed yeast, and the mixture is allowed to stand at 24°C. After 24 hours, when the formation of gas has ceased, the mixture is filtered, cooled to the original temperature, and the specific gravity is then taken. (The urinometer should be standardized.)

Every degree less in specific gravity will indicate 0.23 per cent. of sugar, so that, if the specific gravity of the urine was originally 1032, and after fermentation 1022, the amount of sugar contained would be $10 \times 0.23 = 2.3$ per cent.

This method gives very accurate results when the amount of sugar in the urine is over 0.5 per cent.

Estimation by polarization. 20 to 30 c.c. of urine are placed in a glass beaker, and a small quantity of powdered

plumbic acetate is added; the resulting precipitate is then removed by filtration. The filtered solution is then placed in the glass tube of the polariscope (usually will contain 10 or 20 c.c. of fluid), special care being taken to free the tube of all air-bubbles. The interior of the tube should first be washed with some of the filtered mixture. From the scale attached to the polariscope, better from the average of three different readings of the scale, the percentage of sugar is determined in this way. The deviation to which the tube is directed is read off from the scale as degrees; this number is multiplied by 100, and the result divided by 53.1. If the tube of the polariscope is 20 cm. long, the quotient is divided by 2. (The description and theory of the polariscope may be found in the larger text-books.) Should the urine contain *albumin*, it should be precipitated before the polariscope is used, because albumin deviates light to the left.

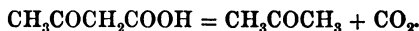
The polarization of urine will give other results from the titration method: (1) if the urine contains other reducing substances besides sugar; (2) if it contains other substances which deviate light to the left, such as oxybutyric acid, which is found in severe cases of diabetes. In such cases it is wise to titrate and to polarize again after the proposed fermentation. Excepting these sources of error, the polarization gives accurate results.

Acetone and aceto-acetic acid. — Both these compounds appear abundantly in the urine after an extensive disintegration of the albuminoids of the body, especially in cases of high fever, in severe anæmias, and in some cases of carcinoma, in hasty consumption, in severe forms of diabetes, and in inanition. In severe diabetes acetonuria occurs even when there has been a total abstinence from the carbohydrates, and when tissue disintegration has not occurred. In addition acetone is found in the urine in cases of disturbances of digestion and in intestinal diseases. In the latter

cases acetone has been found even in the contents of the intestine.

Aceto-acetic acid ($\text{CH}_3\text{COCH}_2\text{COOH}$) is tested by *Gerhardt's* method of reaction to *ferric chloride* solution. On the addition of Fe_2Cl_6 to the urine, a gray precipitate of iron phosphate is thrown down; on the further addition of the ferric chloride, if aceto-acetic acid be present, a deep *claret* color is produced. If the contents of the tube are now shaken, the resulting froth will have a red-violet tinge. On the addition of sulphuric acid the red color will disappear.

On boiling the urine the aceto-acetic acid will be converted into *acetone* and carbonic acid: —



The acetone is distilled over (*distil* about $\frac{1}{2}$ litre of urine with a few drops of hydrochloric acid) and may then be tested by *Lieber's* test, which is conducted as follows: some of the distilled fluid is mixed in a test-tube with a few drops of a solution of iodine in potassium iodide (iodine, 2; potassium iodide, 10; distilled water, 200) and of potassium hydrate solution. Should acetone be present in the urine, an immediate precipitation of yellowish-white characteristically smelling iodoform will result.

Acetone is proved to exist in the urine by means of *Legal's* test, A few drops of a sodium nitro-cyanide solution are added to the urine to be tested and the mixture is then rendered strongly alkaline. A purple-red color changing gradually to a yellow will result; 2 to 3 drops of acetic acid are then added, and, if acetone be present, a color varying from carmine to purplish-red will form where the acid touches the solution. Still, it is doubtful whether acetone occurs preformed in the urine.

The claret-red ferric chloride reaction is of practical diagnostic importance, especially in *diabetes*; the long-continued presence of a large amount of acetone indicates a grave form of the disorder and should suggest a very unfavorable prognosis.

Ehrlich's diazo-reaction. — In many diseases there appear in the urine certain aromatic compounds of whose composition little is known, which unite with *sulpho-diazobenzol* in such a manner as to produce certain characteristic colors.

The chemical course of the *diazo-reaction*: sulphanilic acid ($C_6H_4NH_2SO_3H$), when united with nitrous acid (HNO_2), produces sulpho-diazo-benzol ($C_6H_3NNSO_3H$ [diazo = 2 atoms of N]). The latter body unites with many aromatic amido-compounds to produce colors. In order to produce sulpho-diazo-benzol in a fresh state at any moment, it is necessary to keep a solution of sulphanilic acid in hydrochloric acid, and when it is needed to carry out the test, to add to this solution a solution of sodium nitrite, whereby nitrous acid is formed in a free state which from the sulphanilic acid produces sulpho-diazo-benzol.

Method of conducting the diazo-reaction. Two solutions should be prepared and separately kept, viz.:—

1. Sulphanilic acid,	5.0	2. Sodium nitrite,	0.5
Hydrochloric acid pure, 50.0		Distilled water,	100.0
Distilled water,	1000.0		

In order to carry out the test, 50 c.c. of the sulphanilic acid solution and 1 c.c. of the sodium nitrite solution are mixed in a glass graduate. This mixture is added to the urine which is to be examined, in the proportion of half urine and half mixture, and to these is added an amount of ammonia equal to $\frac{1}{3}$ of the volume of the combined urine and mixture. The entire mixture is then placed in a test-tube and violently shaken. If the *resulting froth* should assume a *deep red* color, the diazo-reaction is positively demonstrated.

The diazo-reaction is still more beautifully shown if we use *para-amido-aceto-phenon* in the proportion of 0.5 to 1000 instead of sulphanilic acid. This constitutes *Friedenwald's* modification of *Ehrlich's* reaction.

The diazo-reaction is obtained in the urine of cases of typhoid fever, pneumonia, measles, miliary tuberculosis, sepsis, and in severe cases of phthisis. It is absent in meningitis. The principal value of the diazo-reaction is its presence in cases of *typhoid fever*, where in doubtful cases it often decides the diagnosis and renders the relapsing character of the subsequent fever positive. The disappearance of the reaction shows that the infection has subsided. In phthisis the appearance of the reaction indicates a bad prognosis.

Fat in the urine is recognized by the milk-like cloudiness of the entire urine, which disappears when some potassium hydrate solution is added to it and the mixture well shaken with ether. Fat is often held in solution in the urine. On microscopical examination numerous small fat globules may be seen. Even on centrifuging the urine the fat may not in all cases be separated. This symptom is called *chyluria*; it represents an individual disease, common in the tropics, and often produced by the *filaria sanguinis* (see Chap. XII.). Chyluria is only seldom the result of occlusion of the thoracic duct; in many cases its etiology is obscure.

Melanin is the coloring matter of melanotic carcinoma, which at times is swept into the circulation and thus appears in the urine, rendering the urine in rare cases a dark black color. In other likewise rare cases an antecedent stage (*melanogen*) of the same coloring matter appears in the urine, from which the latter may be precipitated by ferric chloride.

Sulphuretted hydrogen occurs in rare cases in the urine of patients with cystitis, and is due to the action of certain bacteria which induce a peculiar urinary metamorphosis. H_2S may be recognized by its smell, like that of rotten eggs, or by holding over the urine some paper saturated with plumbic acetate, which will turn brown owing to the formation of plumbic sulphide.

CHEMICAL EXAMINATION OF THE NORMAL ELEMENTS OF THE URINE WHICH ARE QUANTITATIVELY CHANGED BY DISEASE

1. Inorganic Elements

Chlorides occur in the urine chiefly in the shape of sodium chloride; the normal amount depends upon the amount and character of food taken, but averages between 10 to 15 g. of NaCl. They are diminished in fever, especially in *pneumonia* and in inanition.

The test for chlorides is made in this manner: a few drops of acetic acid and a few of a 10 per cent. solution of silver nitrate are added to the urine; a cheesy precipitate will ordinarily be formed,

but in the urine of cases of pneumonia, etc., there will often be only a slight cloudiness. Exact quantitative analysis is made by titration.

Phosphates occur in the urine as potassium sodium, calcium and magnesium phosphates. The quantity daily passed in the urine varies according to the food within wide limits, and is about 3 g. The quantitative analysis for phosphates is by methods of titration and has no essential diagnostic bearing.

Sulphates appear partly as potassium sulphate (preformed sulphuric acid), partly as phenol, indoxyl, and scatoxyl compounds (sulphuric ether compounds).

The separation of these two classes of compounds is performed thus: a few drops of acetic acid and 20 c.c. of a 10 per cent. solution of barium chloride are added to the specimen of urine. By this means the preformed sulphuric acid is precipitated. The solution is then filtered, thus removing the precipitate. In the filtered solution the sulphuric ether compounds will be contained together with the excess of barium chloride. On boiling the filtered solution with hydrochloric acid, the sulphuric ether compounds are split up into phenol and sulphuric acid; the sulphuric acid, uniting with the remaining barium chlorides, is precipitated as barium sulphate.

The test in itself for sulphuric acid has no diagnostic importance; but the quantitative determination of the two forms of sulphuric acid (preformed acid and sulphuric ether) is, because it gives a reliable measure of the intensity of intestinal decomposition. For this purpose the precipitate of barium (baryta) sulphate should be weighed.

Carbonates occur in solution in the urine in essential amounts only after the ingestion of fruit, etc.; they also are combined with special alkalies. Their presence is determined by the evolution of gas resulting from the addition of an acid to the urine. Concerning its diagnostic significance consult under *Reaction*, p. 183.

Ammonia appears in fresh urine, and averages in amount daily between 0.5 and 0.8 g. It is increased in many diseases of the liver and in diabetes to 6 g. Such an in-

crease serves as a guide to determine the severity of a diabetes. The quantitative estimation is made by the addition of chloride of lime to 20 c.c. of urine, to which has also been added 20 c.c. of sulphuric acid of a definitely known composition. The mixture should be prepared in a shallow glass vessel under a bell jar. After 48 hours the change which the sulphuric acid has undergone is measured and from the amount which has been lost the formation of NH_3 is determined. Ammonia is present in large quantities in old *decomposed* (alkaline) urine; its presence may be demonstrated by holding a piece of moistened red litmus paper over a specimen of the urine, or by holding a glass rod which has been dipped in hydrochloric acid over it (cf. p. 183).

Sodium appears in daily quantities of 3 to 6 g. Na_2O ; *potassium*, 2 to 3 g. K_2O . The quantitative estimation of these bodies is performed according to the rules of chemical analysis. Their presence may be of diagnostic value in some cases from the fact that in all cases of extensive albuminoid disintegration, such as occurs in fever and in inanition, the amount of potassium salts is much increased and of the sodium salts much diminished.

2. Organic Elements

Urea (designated often by clinicians with the symbol U^+) is the chief end product of the metabolism of the albuminoids.

Chemical characters. Urea crystallizes in prisms and needles, is soluble in alcohol and in water, insoluble in ether, and forms *biuret* when subjected to dry heat. This latter compound will produce a red color when added to a potassium hydrate solution containing some drops of a solution of cupric sulphate (biuret reaction, see p. 87). By the action of the bacteria in the urine urea becomes transformed into ammonium carbonate, $\text{CO}(\text{ONH}_4)_2$.

It combines with the formation of crystals, with nitric acid, and with oxalic acid.

The normal quantity of urea depends for the most part on the amount of albumin ingested (see Chap. X.); it varies between 20 and 40 g., but is less when little albuminous food has been taken and is physiologically increased after meals rich in such foods.

Pathological *increase* of the amount of urea occurs in fever, in many cases of carcinoma, in anæmia, in leucæmia, in intoxications (phosphorus, arsenic, chloroform, etc., poisoning), and in dyspnoea.

Pathological *diminution* of the amount of the urea excreted occurs in inanition, often in the diffuse diseases of the kidneys (Bright's), and in acute yellow atrophy of the liver.

Qualitative test for urea. — This test is of diagnostic importance in determining the presence of a uræmic condition, in which condition the *vomited matter*, the sputum, the effusions, and the blood may all contain urea. The fluid to be examined for this should be evaporated to the consistency of syrup; the urea is then extracted by alcohol, the solution is filtered, the alcohol is driven off by distillation, the thick liquid remaining is diluted with water, some concentrated nitric acid is added, and a portion examined under the microscope. In a little time the characteristic hexagonal crystals of urea nitrate will be observed.

Quantitative estimation of urea. — This is of great diagnostic value in cases of diseases of metabolism and in dietetics (see Chap. X.). The urea is not determined as much as is the total quantity of nitrogen which appears in the urine.

Approximate Estimation by Titration. Liebig's Method modified by Pflüger

From a graduated burette containing a solution of mercuric nitrate of definite proportions,¹ the fluid is allowed to drop gradually into a test-tube which should contain 10 c.c. of the urine. An insoluble combination of urea nitrate and mercuric oxide is formed.

¹ In 1000 c.c. of the solution there should be 71.48 g. of mercury. The preparation of the solution requires a great deal of care; it may be procured at many chemical factories.

Next to the burette a watch glass with a black bottom should be placed, in which a thick mixture of powdered Na_2CO_3 and water has been prepared. As long as the mercuric nitrate is united with the urea in the urine, it can not react to Na_2CO_3 ; but as soon as all of the urea is used up in the former combination, the $\text{Hg}(\text{NO}_3)_2$ which will now exist in a nascent condition will combine with the Na_2CO_3 to form 2NaNO_3 + carbonic acid (which will arise in bubbles) + yellow HgO . The urine and the mercuric nitrate solution, as it drops into it, should be stirred thoroughly with a glass rod; the thick sodium carbonate mixture should then be stirred with the same rod; as soon as a permanent yellow color appears at the spot of the thick mixture which was touched by the rod, the titration is completed.

The number of cubic centimetres of the mercuric nitrate solution used in the test is multiplied by 0.04 to obtain the percentage of nitrogen in the urine. For example, 21 c.c. of the Hg solution were used in 10 c.c. of urine; $\therefore 21 \times 0.04 = 0.84\%$ of N. If the urine passed in 24 hours happened to be 1500 c.c., then the daily excretion of N would be 12.6 g., which equals 27 g. of urea, because the amount of nitrogen is to the urea as 1 is to 2.143 or $\text{N} : \bar{\text{U}} :: 1 : 2.143$, from which $\bar{\text{U}}$ was determined.

This method is for clinical purposes entirely reliable. A more exact method used requires too many details which must be carefully carried out, such as precipitating the phosphates and sulphates in the urine by a solution of baryta, the reduction of the acidity which arises in the process of titration, the repeated addition of mercuric nitrate solution, etc.

The method now used in all clinical laboratories, one which is entirely reliable, for determining the amount of nitrogen is that first recommended by *Kjeldahl*. It is carried out in this way: 5 c.c. of urine and 20 c.c. of fuming sulphuric acid are placed in a flask, which is then held over a flame until it boils, when the fluid loses its color. The solution is then diluted with 100 c.c. of sodium hydrate whose specific gravity is 1.3 and distilled in a distilling apparatus; 50 or 100 c.c. of a $\frac{1}{10}$ normal acid (sulphuric) is titrated after the distillation with a $\frac{1}{10}$ potassium hydrate solution. (All of the N of the urine is converted into NH_3 , which becomes united with the excess of sulphuric acid into $(\text{NH}_4)_2\text{SO}_4$; the NaHO sets the NH_3 free, and the latter immediately combines in part with the prepared $\frac{1}{10}$ solution of normal sulphuric acid; the part which remains is determined by titration and from it is reck-

oned the entire amount of NH_3 , from which in turn the amount of N contained in the original 5 c.c. of urine is determined).

Example: 5 c.c. of urine were oxidized; 50 c.c. of $\frac{1}{10} \text{H}_2\text{SO}_4$ were prepared; after the distillation 12 c.c. of $\frac{1}{10} \text{NaOH}$ in the titration of the acid remaining free, therefore only 12 c.c. of the $\frac{1}{10} \text{H}_2\text{SO}_4$ remained free; therefore 38 c.c. of the $\frac{1}{10} \text{H}_2\text{SO}_4$ must have been used in combining with the NH_3 which distilled over. For the combination of 38 c.c. of the $\frac{1}{10}$ normal acid, that is, $38 \times 0.049 \text{ g. H}_2\text{SO}_4$, $38 \times 0.0017 \text{ NH}_3$ are necessary; and the amount of nitrogen corresponding would be $38 \times 0.0014 \text{ g.}$, which would be equal to 0.0532 g. of N. Now since this amount was contained in 5 c.c. of urine, the entire percentage in the amount would be 1.064.

Uric acid ($\text{C}_5\text{H}_4\text{N}_4\text{O}_3$) is excreted in the urine, the daily amount being from 0.4 to 1.4 g. The quantity varies in different persons and at different times. The uric acid (together with the xanthin bases) arises from the disintegration of the nuclein which is contained in all cell bodies. The uric acid appears in the urine in solution as neutral sodium urate; in very acid and in very concentrated urine (such as occurs in fever and in congestion of the organs, in cases where a small amount of fluids is taken, and in profuse perspiration) acid sodium urate or pure uric acid is sometimes precipitated when the urine becomes cold (see urinary sediments).

Uric acid excretion is increased in all conditions of increased destruction of leucocytes, in leucæmia (the proportion in the urine is 1 to 16), after the ingestion of food rich in nuclein. In gout the excretion of uric acid varies remarkably.

The qualitative determination of uric acid is sometimes of value in the examination of sediments and concretions (see below), and is performed by means of the *murexide* test, which is the following:—

To the specimen to be examined 3 or 4 drops of concentrated nitric acid are added in a porcelain dish and the contents of the dish evaporated to dryness; if uric acid be present in the specimen, an orange-yellow color will be produced, which will be changed by the addition of ammonia to be a purple-red.

The qualitative examination for its presence in the blood is carried out as was suggested by Garrod with a thread, constituting *Garrod's thread test*. The method is as follows: 10 c.c. of blood are obtained by wet cupping; the blood is permitted to coagulate and to settle in the cup, the serum is transferred into a watch glass, rendered acid with some 30 per cent. acetic acid, and in the mixt-

ure a thin linen thread is placed. The glass should remain covered for 24 hours, when it is examined under a low power of the microscope; should uric acid have been present, many crystals of it will be found adhering to the thread. Its presence occurs in attacks of gout and in nephritis.

To estimate uric acid quantitatively, the urine should be mixed with magnesia and silver nitrate; the resulting urates of magnesia and silver are then combined with sulphuretted hydrogen and the amount of uric acid determined by weighing. For an exact description consult the various text-books on chemical analysis.

Oxalic acid ($\text{COOH} \cdot \text{COOH}$) is excreted to the amount of 0.02 g. in the 24 hours, either in solution or as a sediment of calcium oxalate (see below).

Xanthin bases (xanthin $\text{C}_5\text{H}_4\text{N}_4\text{O}_9$, hypoxanthin $\text{C}_5\text{H}_4\text{N}_4\text{O}$) appear in small quantities in the urine and result, like uric acid, from the disintegration of nuclein substances. The sum total of uric acid and xanthin bases are designated as *alloxan bodies*. Consult text-books for the quantitative determination. The alloxan bodies are increased in all conditions which involve augmented disintegration of leucocytes.

Kreatinin ($\text{C}_4\text{H}_7\text{N}_3\text{O}$), daily quantity from 0.6 to 1.3 g., is without diagnostic importance, as is

Hippuric acid ($\text{C}_9\text{H}_9\text{NO}_3$), whose daily amount of excretion is between 0.25 and 0.5 g., and which is formed from benzoic acid ($\text{C}_6\text{H}_5\text{COOH}$) and glycol ($\text{CH}_2\text{NH}_2\text{COOH}$).

Indican ($\text{C}_8\text{H}_8\text{NKSO}_4$), potassium indoxyl sulphate, present in small quantities in the urine, is increased in cases of extensive decomposition in the intestinal tract, therefore in all diseases of the abdominal cavity which tend to diminish peristalsis and hinder absorption, especially in *peritonitis* and *intestinal obstruction*; at the same time the amount of indican is greater the higher the obstruction is situated. Obstruction of the large intestine gives rise to but little indican. Indican is also increased in cases of putrid supuration.

Chemical attributes of indican. From the decomposition of albuminoids in the intestine or in foci of suppurative *indol* ($\text{C}_8\text{H}_7\text{N}$) is produced; indol is oxidized in the organism into *indoxyl*; like

all aromatic compounds, indoxyl combines with sulphuric acid. The test for indican is based on the development of indigo blue.

Test for indican in the urine. The specimen of urine is mixed with an equal volume of hydrochloric acid; fresh chloride of lime solution is then added drop by drop while the mixture is being stirred. The chloride of lime solution should be composed of 5 parts of chlorinated lime and 1000 parts of distilled water. If indican be abundantly present, the urine will turn to a blue color, or a precipitate of indigo in the shape of blue flakes will take place. By the addition of ether or chloroform the indigo blue will be removed. Very dark urine should, before the test is applied, be mixed with some plumbic acetate and then filtered.

Indigo red is demonstrated by boiling the urine and then adding some nitric acid drop by drop, while carefully continuing the boiling process until a deep-red color appears. Should the mixture then be shaken, the froth will assume a blue-violet color, which is at once removed by the addition of chloroform or ether. This constitutes *Rosenbach's reaction*. The composition of indigo red is not as yet known; this reaction occurs quite in parallelism with the indigo blue reaction in the urine of cases of severe intestinal affections, although one or the other may be demonstrated in mild cases.

Phenols: C_6H_5OH is carbolic acid or phenol; $C_6H_4CH_3OH$ is cresol; they occur in the urine combined with sulphuric acid (sulphuric ether). In the normal state 0.017 to 0.05 g. of phenols are excreted; increase in this amount to 0.06 g. occurs in cases of decomposition occurring within the organism, in which cases the excretion of phenols offers a diagnostic measure of great value in determining the intensity of the process.

Test for phenol: 200 c.c. of urine are mixed with 40 c.c. of hydrochloric acid and distilled until 150 c.c. have gone over into the receiver; an aqueous solution of bromine is added to the distilled portion until it assumes a yellow color. A precipitate will be formed if phenol be present. The precipitate is tribromophenol. By weighing this the amount of phenol in the entire urine may be determined.

EXAMINATION OF THE URINARY SEDIMENTS

If the urine be very cloudy, or if it contain a sediment, pour it into a conical glass and permit the deposit to collect for several hours; then pour off the superambient fluid and place a specimen of the sediment upon the slide for microscopical examination.

For the speedy collection of a scanty sediment use a *sedimentator* (*centrifuge apparatus*).

Before using the microscope, test the reaction of the urine and heat a sample so as to gain some knowledge as to its constituents. For if the urine be acid and cloudy and the cloudiness disappears on heating, the sediment will be recognized as composed of urates chiefly. Should the urine be acid and the cloudiness do not disappear on heating, but on the addition of potassium hydrate, then the sediment is due to uric acid. If the urine be alkaline and cloudy and the cloudiness disappeared on the addition of hydrochloric acid with or without the evolution of gas, then it may be considered that the sediment was composed of lime carbonate or phosphate.

UNORGANIZED SEDIMENTS

In Acid Urine

Acid sodium urates (Fig. 35) are amorphous granules collected in clusters, usually colored yellow. They form the *brick-dust deposit* (*sedimentum lateritium*), are soluble on heating as well as on the addition of potassium hydrate. On adding some hydrochloric acid to a specimen under the microscope, crystallization of uric acid may be observed. They are of no diagnostic importance, and indicate only the acid condition and concentration of the urine.

Uric acid (Fig. 35) occurs in the shape of whetstones, and in cylinders (also in the shape of spears and rosettes) chiefly of a yellow color; it dissolves on the addition of potassium hydrate, but not on heating. It is recognized outside of the shape of its crystals by the murexide reaction (see p. 207). An abundant deposit of uric acid does not in itself denote that the uric acid is increased, but often only that the con-

ions of solubility are unfavorable, either because there is too small an amount of water, or because there is too much acid in the urine; still, when abundantly present, it should demand a quantitative analysis. An abundant uric acid deposit in the urine indicates the presence of the so-called uric acid diathesis (*nephritis urica*, *arthritis urica*).



FIG. 35. — URINARY DEPOSITS IN ACID URINE.

Oxalate of lime (Fig. 35) (the reaction of the urine is usually neutral) forms in octahedral crystals. If present only as solitary crystals, it has no significance; even an abundance of them in the urinary deposit does not always indicate an increase in the excretion of oxalic acid; to determine this a quantitative analysis is necessary.

Cystin occurs rarely as a deposit. When it does it is pathognomonic of a special form of metabolic disturbance (see Chap. X.). It consists of hexagonal crystals which are readily soluble in ammonia.

Leucin (amido-capronic acid) (Fig. 36) and *tyrosin* (amido-paracumaric acid) are likewise rarely found as urinary deposits; they occur in the urine in cases of acute yellow atrophy of the liver and in poisoning by phosphorus. *Leucin* crystallizes in yellow-white crystals which are often in the shape of striped radiatory globules; *tyrosin* crystallizes as beautiful needle-like bundles.

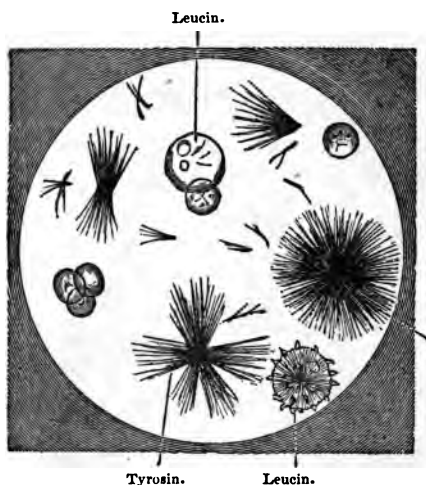


FIG. 36. — SEDIMENT OF URINE IN ACUTE YELLOW ATROPHY OF THE LIVER.

In Alkaline Urine (Fig. 37)

The *ammonio-magnesia phosphates* or *triple phosphates* ($\text{NH}_4\text{MgPO}_4 + 6\text{H}_2\text{O}$) crystallize in coffin-like crystals which are very readily soluble in acetic acid.

Phosphate of lime occurs either as $\text{Ca}_3(\text{PO}_4)_2$ in the form of irregular granules, or as CaHPO_4 in cuneiform crystals, which are usually collected together in masses and often in rosettes.

Carbonate of lime (CaCO_3) occurs as round regular granules or in the shape of dumb-bells, and dissolves on the addition of an acid with an evolution of gas.

Ammonium urate occurs in the shape of pine cones, or in irregular fusiform crystals.

The alkaline deposits, outside of indicating the reaction of the urine (see above), have no farther diagnostic significance.

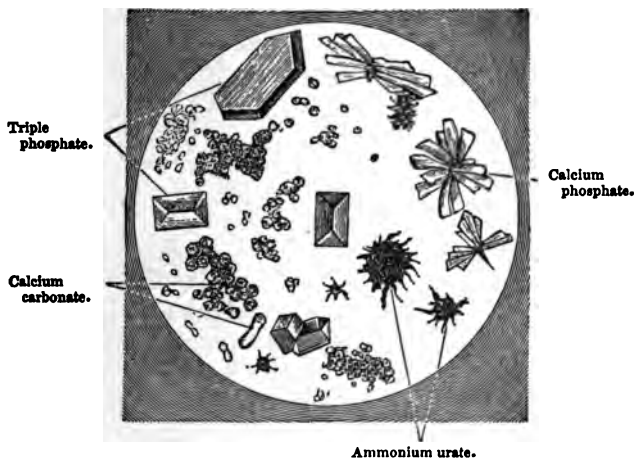


FIG. 37. — SEDIMENT OF AMMONIACAL URINE.

ORGANIZED SEDIMENTS (Figs. 38 and 39)

These are of the utmost importance in the diagnosis of diseases of the kidneys (see Chap. IX.).

White blood corpuscles (leucocytes) appear sparsely in the urine of healthy persons; should they be abundant, however, it indicates an inflammation or a suppuration in some spot along the genito-urinary tract, a nephritis, pyelitis, cystitis, gonorrhœa, or a leucorrhœa in women.

Red blood corpuscles occur mostly as pale corpuscles, owing to imbibition of water; they indicate the presence of a hæmorrhage somewhere in the genito-urinary apparatus (see hæmaturia, p. 189).

Renal epithelium (Fig. 38) occur as round or cubical cells containing nuclei, and indicate chiefly an affection of the kidney. They often coalesce into *epithelial casts*. *Fatty epithelial cells* (masses of fat granules) are of the utmost diagnostic importance (Fig. 39); they indicate chronic parenchymatous nephritis in the 2d stage of a fatty degeneration.

Epithelium from the renal pelvis, of the *ureters*, and of the *bladder* can not be distinguished from each other; they occur either as flat polygonal, or more or less round cells, provided with processes and in part containing nuclei. An abundance of these cells in the urine justifies the diagnosis of a pyelitis, of a cystitis, or of an inflammation of the ureter (see differential diagnosis, Chap. IX.).

Epithelium from the vagina consists of large flat epithelium like the buccal epithelium; *epithelium from the male urethra* is of the cylindrical variety, and appears sometimes in gonorrhœal pus.

Casts in the urine are probably exudations from the renal tubules. They appear as:—

1. *Hyaline casts*, which are small, transparent, homogeneous formations, with not very definite contours. Their presence is not a proof of nephritis. They occur also in congestion, in fever, and in jaundice, and even in very small quantities in the healthy person.
2. *Epithelial casts*, consisting of agglutinated epithelial cells, indicate the presence of a nephritis; they are often deformed, more or less granular (*granular casts*), and often are covered by fatty epithelial cells.
3. *Blood casts* occur only in hæmorrhage of the kidneys.
4. *Waxy casts* have sharp outlines, a yellow glistening appearance, and occur only in chronic nephritis.
5. *Brown casts* occur rarely in severe infectious diseases, and in fractures of bones.

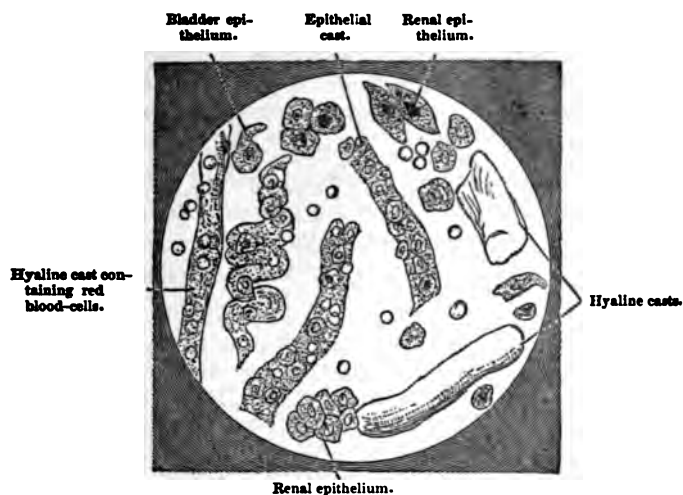


FIG. 88.—SEDIMENT IN ACUTE NEPHRITIS.

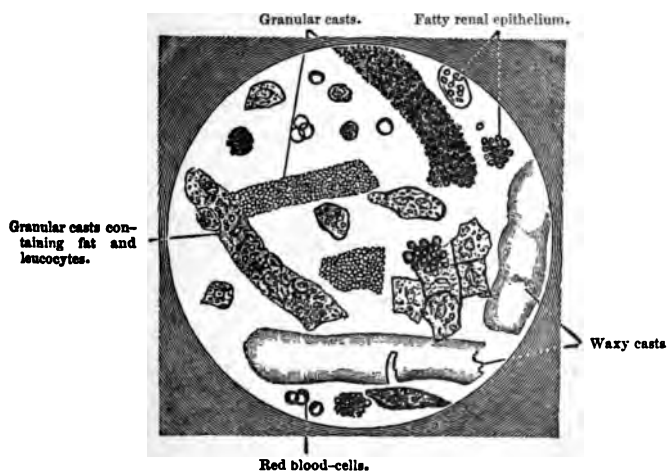


FIG. 89.—SEDIMENT IN CHRONIC NEPHRITIS.

Cylindrical formations which look like casts are often seen; they are produced by accumulations of bacteria or of amorphous urates.

Micro-organisms may occur in the urine in many infectious diseases (diphtheria, relapsing fever, and in typhoid fever). The presence of gonococci and of *tubercle* bacilli have of course a diagnostic significance. In cystitis and in pyelonephritis an abundance of microbes appear in the freshly voided urine. (For their demonstration, see Chap. XII.)

Parasites have been observed in rare cases in the urine in the shape of particles of *echinococcus*, embryos of *Filaria sanguinis*, which, like the *Distoma hæmatobium*, have given rise to hæmaturia.

TESTS FOR CERTAIN HETEROGENEOUS SUBSTANCES OCCURRING IN THE URINE

The determination of the presence of foreign substances in the urine is of importance in diagnosing the various *intoxications*. Irrespective of this, it is often of interest from a therapeutic standpoint whether a substance has been absorbed by the organism. Finally, by demonstrating the presence of a medicine in the urine, we may be able to form opinions concerning the patient's statements.

Iodine appears in the urine after the use of potassium iodide and iodoform. Add to the urine a few drops of fuming nitric acid, or of an aqueous solution of chlorine, and a few cubic centimetres of chloroform; then shake the test-tube containing the mixture; should iodine be present, the chloroform will assume a red-violet color.

Bromine appears in the urine after the use of the bromides. Test in the same manner as for iodine. When large quantities are present, the chloroform will become yellow. The test will not detect a small quantity. In the latter case, we render the urine alkaline with sodium carbonate, and add to it 2 g. of potassium nitrate. Pour the mixture then into a platinum dish, and evaporate and heat the dry residue until it melts. The residue,

when cooled, is dissolved in water rendered strongly acid with hydrochloric acid, and is then shaken with chloroform. With this test, if the urine contains only a very small quantity of bromine, the chloroform will become yellow.

Iron. — Urine containing much iron will become greenish-black on the addition of ammonium sulphide. To test the presence of only a small quantity of iron, 50 c.c. of urine are evaporated in a platinum dish, the dry residue is converted into an ash, and then the ash is subjected to the action of a weak solution of hydrochloric acid. Should iron be present, a blue precipitate will result after the addition of some potassium ferrocyanide.

Arsenic. — In order to successfully carry out the test for arsenic, the organic substances in the urine must be removed. This may be done as follows: one or two litres of urine are evaporated in a porcelain dish to $\frac{1}{4}$ of its volume; then an equal volume of concentrated hydrochloric acid is added to the residue and the mixture covered and placed on a water bath. At regular intervals during the heating on the bath, 2 or 3 g. potassium chlorate are added, until the mixture becomes entirely light yellow. It is then rapidly heated and boiled until all smell of chlorine disappears, when it is strongly diluted with water. For many hours after this, sulphuretted hydrogen is passed through the solution, producing a precipitate of arsenic sulphide, which is removed by filtration, dried, dissolved in a small dish by a mixture of a few drops of nitric and sulphuric acids, heated until the acid smell has disappeared, then strongly diluted with water, and the remaining solution is then subjected to the *Marsh mirror test* for arsenic.

The *Marsh test* is performed in the simplest way, as follows: a test-tube is provided with a cork in which a hole has been bored; into the hole one end of a glass tube — which has been bent to a desirable angle, and whose other extremity has been drawn out to a capillary point — is introduced. The test-tube is now filled with zinc and dilute sulphuric acid, and with some of the previously prepared solution of urine. Gas will be immediately evolved, when the cork and attached glass tube should be introduced into the mouth of the test-tube. When the gas is streaming off strongly, the gas at the capillary end of the tube should be ignited by a match. If arsenic be present in the solution, the gas will burn with a blue flame, and if a clean, dry, porcelain plate be held over the flame, a mirror of deposited arsenic will form on the plate. This mirror has a metallic, glistening appearance, and will be dissolved by a solution of sodium hypochlorite.

Lead. — The organic substances should be disintegrated by hydrochloric acid and potassium chlorate, as was done in the test for arsenic (see above), and, through the diluted mixture, which has been thereby rendered weakly acid, sulphuretted hydrogen is permitted to pass; this will form a precipitate of the brownish-black plumbic sulphide when lead is present.

Mercury. — About 1 litre of urine is heated to 60° to 80° C., made acid by the addition of hydrochloric acid, and then thoroughly oxidized by the addition of fine copper filings (lametta). After an hour, the urine is decanted; the copper filings are washed with hot water, then with alcohol, and finally with ether, and thoroughly dried between layers of filter paper. The filings are then placed in a small glass tube, which is thereafter drawn out at both ends into capillary points. The tube is then heated in the flame, the mercury becomes sublimed, and in the capillary ends metallic, silver-like rings will have formed. If a small piece of iodine has been put in the tube before the ends of the tube have been drawn out, the red mercuric iodide would be formed instead.

Carbolic acid. — If much carbolic has been taken, the urine will have a greenish-brown appearance, and by exposure to the air it will become darker. The same color results from the ingestion of *hydroquinon* and from *uva ursi*. The test for carbolic acid is made by adding bromine water to the distilled mixture (see p. 209), or by the test for the presence of the sulphuric ethers (see p. 203).

Quinine. — Shake well a large quantity of urine, to which some ammonia has been added, with ether. This will take up the quinine. The residue left after evaporating the ether is mixed with acidulated water. The mixture is then subjected to the action of chlorine water, and after that to ammonia. A green color will develop if quinine be present.

Salicylic acid ($C_6H_4OH \cdot COOH$). — The urine will give a blue-violet color if ferric chloride be added. Should the reaction to this be negative, acidulate in a graduate 30 c.c. of urine with sulphuric acid, and add to it 30 c.c. of ether; then shake the mixture thoroughly, pour off the ether, and to the latter add some of a ferric chloride solution drop by drop. A blue color will ensue even where the quantity of salicylic acid present is very small.

Antipyrin. — A red color is produced by the addition of ferric chloride to the urine.

Antifebrin. — A red color is produced when the urine is boiled with hydrochloric acid and afterwards mixed with some of a 3 per

cent. carbolic solution and with ferric chloride; on the addition of ammonia, it changes to a blue color.

Phenacetin.—A brownish-red color will ensue on the addition of ferric chloride.

Tannin.—With ferric chloride the urine becomes dark blue, almost black.

Napthalin.—After the ingestion of large doses, the urine will be turned to a green color by the addition of concentrated sulphuric acid.

Turpentine.—The urine has an odor of violets.

Rhubarb and senna (chrysophanic acid).—The urine becomes purple-red on the addition of sodium hydrate. Sodium carbonate will also produce the same reaction.

Santonin.—The urine, when santonin has been taken, has a straw color, which is turned to red by the addition of sodium hydrate. Sodium carbonate, however, will *not* produce the red color.

CHAPTER IX

DIAGNOSIS OF THE DISEASES OF THE KIDNEYS

THE DIFFUSE DISEASES OF THE KIDNEYS (BRIGHT'S DISEASE)

THE diffuse diseases of the kidneys are recognized by the simultaneous presence of *œdema* and *albuminuria*; they are classified under the head of *Bright's disease*. The specific diagnosis of the form of Bright's disease present is decided chiefly by the examination of the urine (see Chap. VIII.), and, in addition to this, by the history, the course of the disease, and by the examination of the other organs (heart, vessels, liver, spleen, and the eyes).

In reference to the *history*, the following data are especially important: alcoholism often leads to the production of a chronic nephritis; chronic intoxications and gout lead frequently to the primary atrophy of the kidney; severe exposure, frequent drenching of the skin, the action of the toxic agents, especially the acute infectious diseases (scarlatina, etc.), are potent in producing acute nephritis. *Tertiary syphilis, suppuration, phthisis, and malaria may lead to amyloid degeneration of the kidney.* Inquiry should always be made as to the duration and course of the disease, and the investigation should elicit whether there have been previously any nephritic symptoms, such as *œdema*, urinary changes, headaches, vomiting, asthma, and visual disturbances.

Concerning *œdema*, see p. 10. For the tests for albumin, see p. 186.

In many periods and in many forms of Bright's disease either *œdema* or *albuminuria* may be absent, and not alone this, but in some rare cases both may for a time be wanting. Under these

circumstances the consideration of the course of the disease, the examination of the heart (cardiac hypertrophy in atropic kidney) and of the pulse, and often the large quantities of urine will aid the diagnosis. In spite of these exceptions it is wise to establish the rule that œdema and albuminuria are the cardinal symptoms of diffuse diseases of the kidneys.

In order to make a diagnosis of the form of Bright's disease present in each case, it is best to fix in the mind a systematized scheme of the various forms of the disease which occur.

BRIGHT'S DISEASE

ŒDEMA WITH ALBUMINURIA

	INFLAMMATORY FORM	NON-INFLAMMATORY FORM
First stage . .	Acute hæmorrhagic nephritis.	Œdema from venous stasis (chronic congested kidney).
Second stage .	Chronic nephritis; fatty degeneration.	Amyloid degeneration.
Third stage . .	Secondary atrophy; white atrophic kidney.	Primary atrophy (red atrophic kidney; arterio-sclerotic, lead, and gout kidneys).

In a class by themselves are the œdema and albuminuria of pregnancy — the kidneys of pregnancy.

In the first column of this table or scheme the forms of nephritis are mentioned, as well as how they sometimes develop in order from one form to the other; still the chronic form may occur *primarily* without there having been an acute inflammation before its development.

Clinical diagnosis must renounce deciding between the presence of an *interstitial* and a *parenchymatous* inflammation, because the symptoms do not in most respects harmonize with the pathological anatomical processes. Still the proof of the existence of fat in the urinary deposit is

a positive indication of a parenchymatous process, while, on the other hand, the absence of an essential sediment in the urine would point to an interstitial inflammation.

In the second column those forms are mentioned which exist individually and which are *not related to each other*, but which in their clinical features very much resemble the forms adjoining them in the first column.

The chronic congested kidney strictly should not belong to this group, but clinically it gives a symptom picture so closely resembling the essential forms of Bright's disease that it appeared to the author justifiable to place it in the table.

CHIEF SYMPTOMS OF THE VARIOUS FORMS OF BRIGHT'S DISEASE

Acute hæmorrhagic nephritis presents the following clinical features: generally severe anasarca, and especially oedema of the face. The urine contains much albumin and a good deal of blood. Its quantity is much diminished, and its specific gravity is high. The sediment will be found to consist of red blood corpuscles, hyaline and granular casts as well as blood and epithelial cells.

The diagnosis should include an inquiry into the etiological factor: is it a nephritis due to an infection, to a poison, or to exposure? or is it an exacerbation of the chronic form? The prognosis is essentially influenced by the quantity of urine and the presence of uræmic symptoms (headache, vomiting, coma, and convulsions).

Chronic nephritis, fatty degeneration.— Usually extensive oedema, accompanied by a large amount of albumin. The quantity of urine varies. The urinary sediment is characteristic and contains *globules of fatty granules*, granular and often waxy casts, and much epithelium. The disease is *usually fatal* in from one to two years, in an attack of uræ-

mia or through complications (rupture of the œdematous skin and erysipelas, pneumonia, etc.).

Primary atrophic kidney. — Abundant urine of low specific gravity, greenish-yellow of color, with little albumin, very little or no sediment. *Hypertrophy of the heart*, pulse of very high tension, retinitis frequent; begins insidiously and often develops very slowly. It occurs in general arteriosclerosis (arterio-capillary fibrosis), in gout, and in plumbism (uric acid kidney).

The atrophic kidney may also develop from a previous nephritis of long standing or from a puerperal nephritis. Rarely, it may arise from chronic congested kidney. The development of a secondary atrophic kidney from a chronic parenchymatous nephritis is exceedingly rare.

Chronic congestion of the kidney presents œdema, chiefly in the legs, cyanosis, and dyspnœa. There is a coexisting cardiac or pulmonary affection. The urine, scanty and dark-colored, is of a high specific gravity. It shows the brick-dust deposit (*sedimentum lateritium*), and contains but little albumin.

Amyloid degeneration of the kidney proceeds generally with the symptoms of a chronic nephritis. The proof of the etiological factor is necessary to a diagnosis (see "history"), as is also the simultaneous enlargement of the liver and spleen.

The other diseases of the kidney are not accompanied with œdema, but often with changes occurring in the urine. They are recognized partly by these features and partly by the occurrence of pains in the region of the kidneys, by palpation and percussion of the abdomen and especially of the renal region.

The following are chiefly to be considered: hæmorrhagic infarction of the liver, pyelonephritis, renal calculi, renal tuberculosis, tumors of the kidney, and movable kidney.

Pains in the region of the kidneys (in the loins) appear so frequently in entirely different diseases that this symptom should be used only with reserve in making a diagnosis of kidney disease. Paroxysmal severe pain (*renal colic*) is the sign of the presence of renal calculi.

SITUATION OF THE KIDNEYS, PERCUSSION

The kidneys lie between the 12th dorsal and 3d lumbar vertebræ. The right kidney reaches to the liver above, the left to the spleen.

The object of percussion of the renal region is to locate the lower and outer borders of the kidneys; however, the results of this are not entirely reliable, on account of the large fat accumulation which sometimes envelops the capsule of the kidneys and on account of the varying condition of the intestines as to solid contents.

Marked increase of renal dulness occurs when the kidney is enlarged by the development of a tumor of the organ (see below); the entire absence of renal dulness justifies the conclusion that a movable kidney (floating kidney) is present, which is found much more frequently on the right side than on the left.

CHIEF SYMPTOMS OF A FEW OF THE NON-DIFFUSE DISEASES OF THE KIDNEYS

Hæmorrhagic infarct of the kidney is characterized by the sudden appearance of *hæmaturia*; pain in the region of the kidney and a slight rise in temperature. The diagnosis is aided by the proof of the etiological factor, an embolism due to a valvular defect or cardiac dilatation. The *hæmaturia* disappears quickly.

Suppurative nephritis (pyelonephritis).—There is an irregular remittent type of fever associated with chills; the urinary sediment contains pure *pus*, usually without casts; there is blood in the urine only when the pyelonephritis has been caused by trauma or renal calculus. Severe pain in the renal region is frequent.

Tumor of the kidney is determined by palpation, bimanual palpation being often necessary; it is often very difficult to determine the renal origin of the tumor, the diagnosis being aided by the immobility of the tumor on forced respiration, the constriction of the colon, in certain cases by the superposition of the distended large intestine over the tumor, and the increased area of renal dulness.

We must decide differentially between an echinococcus (positively determined when the hooklets or the cyst wall is found in the fluid aspirated from the tumor), hydronephrosis (determined by the intermittent filling and emptying of the fluctuating sac and by the presence of urea in the fluid gained by aspiration), and carcinoma and sarcoma (they are solid tumors and are accompanied by a rapidly progressing cachexia).

Renal calculi (nephrolithiasis) is characterized by attacks of renal colic which terminate with the passage of the concretion; by the excretion of sediment independent of the attack of colic, the sediment being chiefly composed of phosphates or urates; by the frequent occurrence of hæmaturia. The reaction of the urine varies according to the nature of the calculi.

Movable kidney.—On palpation a movable abdominal tumor of the shape of the kidney is felt. The area of kidney dulness is obliterated. There are numerous nervous complaints, the patient complaining especially of traction and weight in the abdomen.

Bladder diseases present for diagnosis these conspicuous forms:—

Cystitis, which is diagnosed by the presence of frequent, painful vesical tenesmus, associated with the voiding of dirty cloudy urine full of pus cells and frequently the seat of ammoniacal decomposition (see pp. 185 and 212). The

cause of the cystitis must be determined (gonorrhœa, urethral stricture, hypertrophy of the prostate, stone in the bladder, *paralysis of the bladder from spinal cord disease*). In acute cystitis (the result of exposure or of gonorrhœa) the urine is scanty, cloudy, acid in reaction, bloody, and there is intense local pain and spasm.

Stone in the bladder is diagnosed by the presence of frequent hæmaturia without a characteristic sediment, disturbances in passing urine, in many cases an accompanying cystitis and pains which radiate to the end of the penis. The diagnosis is only positively made by sounding the bladder.

Tumors of the bladder (papilloma [papillomatous fibroma, villous disease] and carcinoma) are diagnosed by the occurrence of attacks of pain and hæmorrhages in the bladder, and by the presence of a chronic cystitis. The diagnosis is only established where a tumor can be felt by a digital examination in the rectum or vagina, or by the catheter introduced into the bladder, or where particles of the tumor can be demonstrated to exist in the urine. Finally the *endoscope* or *cystoscope* may show the presence of a growth.

THE EXAMINATION OF RENAL AND VESICAL CALCULI

We distinguished between: 1. *Uric acid* calculi, which are most frequent in occurrence; they are hard, their surface is smooth or only a little nodular, their color varies from a yellow to a reddish-brown. Calculi composed of *ammonium urate* are brittle, and of a dirty grayish-yellow color. 2. *Oxalate of lime* calculi (mulberry calculi) are very hard, they have a rough warty surface, and vary in color from brown to black. 3. *Phosphatic* calculi (composed of calcium phosphate and of ammonio-magnesium phosphates) are soft and easily pulverized; their surface is sandy and

rough, often glistening; their color is usually white. 4. *Calculi* formed of the *carbonates* are hard, like chalk, with a smooth surface and of a white color. 5. *Cystin* calculi are as a rule small, tolerably hard, smooth, and yellowish. 6. *Xanthin* calculi are quite hard, of a cinnamon-brown color, and their surface when rubbed glistens like wax.

Frequently the concretion or stone is not composed of one material alone, but the nucleus may be composed of one chemical compound and the body of the calculus of another.

Their composition is determined by a chemical analysis like the following, which is recommended by *Salkowski*.

It is advisable for conducting the analysis to determine whether the calculus is composed of organic or of inorganic materials. If it be of an organic compound, the calculus when pulverized will be completely consumed when heated in a platinum capsule or it leaves but a very little ash residue. In this case the calculus is composed of uric acid, of ammonium urate, of cystin, or of xanthin. Should an ash residue be left after heating, the calculus may be composed of urates, phosphates, or oxalates.

I. The Uric Acid Calculus is Completely Consumed by Heat

The powdered calculus is digested with a weak solution of hydrochloric acid and gentle heat.

a. The portion of powdered calculus used will either be dissolved completely or for the greatest part; in this case the calculus is composed of cystin or xanthin. *Cystin* will be dissolved by ammonia and will crystallize after the evaporation of the solution into hexagonal crystals.

Xanthin is tested by dissolving a portion of the powdered calculus in a porcelain dish with some nitric acid and slowly evaporating the mixture. If xanthin be present, a lemon-yellow residue will be left which is *not* soluble in ammonia, but which will change to a reddish-yellow on the addition of some potassium hydrate.

b. Or the portion used will not be dissolved completely, in which case the solution is filtered, and the filtrate is tested for *uric acid* by the murexide test; the filtrate may contain ammonium chloride, which may be tested by adding some sodium bicarbonate and heating the mixture, when the odor of *ammonia* will be

detected; or some moistened red litmus paper may be held over the dish containing the mixture and the red litmus paper will become blue; or a glass rod dipped into hydrochloric acid may be held over the dish and a cloud will form above the dish about the rod.

II. The Calculus may turn Black, but may not be Consumed

Another portion of the pulverized stone is digested with some weak hydrochloric acid and exposed to heat. Should the powdered stone effervesce, it contains a *carbonate*.

a. Complete solution of the contents of the dish denotes an absence of uric acid.

b. Incomplete solution denotes that the residue may contain uric acid or an albuminoid substance. This may be determined by the murexide test.

The filtered solution is rendered slightly alkaline by the addition of ammonia; it is then rendered slightly acid again with acetic acid. Should a white powdery precipitate form, it denotes the presence of *oxalate of lime*. This is removed by filtration, and the filtered solution should be tested for phosphoric acid, for calcium, and magnesium. A portion of the filtered solution is mixed with some ferric chloride, which, if *phosphoric acid* be present, will produce a grayish-white precipitate. The chief remnant of the filtered solution should then be mixed with ammonium oxalate, whereupon a precipitate being formed would denote the presence of *magnesia*. The lime is removed by filtration and to the filtered solution some of a solution of sodium phosphate is added and the whole subjected to heat. It is then rendered alkaline with ammonia. A slowly developing crystallizing deposit will indicate the presence of *magnesia*.

CHAPTER X

THE DIAGNOSIS OF THE DISORDERS OF METABOLISM

LAWS OF NORMAL METABOLIC PROCESSES

THE human body requires the ingestion of food material in order that, on the one hand, it may conduct its vital functions undisturbed, and on the other hand, that it may not consume the essential constituents of its own tissues, which consist of the albuminoids, fats, carbohydrates, water, and inorganic salts.

The products of the disintegration of the albuminoids are excreted by the urine as urea, uric acid, etc.; the fats and carbohydrates are oxidized into carbonic acid and removed from the body in the breath, but they are nevertheless of great importance in the preservation of the organism.

The albuminoids are very complex bodies, whose chemical composition has not as yet been sufficiently investigated; only this much is known, that from the disintegration of albumin occurring in the body different chemical groups are formed, viz.: (1) a nitrogenous, urea-like group, which is excreted in the urine; (2) an aromatic group containing or represented by C_6H_5 , which is likewise excreted in the urine; (3) a group resembling the fats, and which further *act like fats and carbohydrates* and are removed from the body by the breath as CO_2 . In this way the positive fact is explained that *fat and sugar can be formed in the body from the albumins*, and that albumin, when introduced in sufficient quantity, can replace the other nutritive materials. On the other hand, the fats and carbohydrates, which lack the nitrogenous and aromatic compounds, can only replace the albumin to a limited extent.

The requisite amount of food-stuffs. — In order that the nutrition of the body may be kept at the normal, it requires that the body be given a certain *amount* of food. In order that in estimating the amount of nourishment needed, a uniform *measure* may be used for the different food-stuffs, we make use of the *amount of heat* which is developed from the disintegration of food, and which has been used by different investigators in their experiments and investigations. Therefore as the unit value we make use of the *calorie*, which is that amount or quantity of heat which is necessary to raise one kilogramme of water one degree in temperature.

We may therefore substitute for the amount of food-stuff its corresponding value in calories, in this manner : —

1 g. albumin	= 4.1 calories
1 g. fat	= 9.3 calories
1 g. carbohydrate	= 4.1 calories
1 g. alcohol	= 7.0 calories

Instead of saying a strong man needs for nourishment 118 g. of albumin, 56 g. of fat, and 500 g. of carbohydrates, we may say he needs the equivalent of 3054.6 calories.

The number of calories which are needed for nourishment by a healthy man depends on his body weight, on the amount of work performed by him, and on his previous condition of nutrition. A healthy, strong laborer weighing about 70 kg. requires daily about 3000 calories, and when doing work requiring the exercise of additional effort, the necessary number of calories will rise to between 4000 and 5000; a less strong laborer, whose weight is about 50 kg., requires about 2400 calories. The requisite amount therefore for a healthy man is about 45 calories for every kilogramme of weight. However, it is entirely improper, if we wish to determine the amount of nourishment needed by a healthy man, to simply multiply his body weight by a cer-

tain number of calories. Moreover, the necessary number of calories necessary for his sustenance depends essentially on the condition of the physical changes of the days immediately preceding. For instance, if a man, otherwise normal, have an œsophageal stricture of long standing which has left him badly nourished, and by reason of this much emaciated, he may retain the condition in which he is at the time, even with from 1000 to 1500 calories, yes, even with less. To estimate the number of calories necessary, it is essential to study in each individual case the nutrition of the body and conditions of the preceding days.

Relations of the food-stuffs to each other. — It is very essential to observe in nutrition that the representation of the various food-stuffs corresponding to their caloric values is *only possible up to certain limits*. It is, moreover, necessary that a definite amount of *albumin* be always administered to the body; *this can not be replaced by fats and carbohydrates*. The quantity of this necessary amount of albumin (*albumin necessary to nutrition*) depends upon the condition of nutrition, rather on the store of albumin in the body, and, on the other hand, upon the amount of carbohydrates and fats which have at the same time been administered.

The amount of albumin necessary for the nutrition of a strong, well-nourished man is from 80 to 100 g.; in the poorly nourished and in the non-worker it may be less.

It is only when the amount of albumin necessary for the body's nutrition has been reached, that the food-stuffs may be reckoned entirely according to their corresponding caloric values; and it depends more upon the condition of the stomach and the digestion whether fats and carbohydrates or whether more albumin should be given.

Before the power of substitution of one food for another, by virtue of the measurement of its calories, was sharply defined, it was known that the various foods could replace each other under

certain conditions; these were called *isodynams*. Thus 100 g. of fat equalled 211 g. of albumin, or 232 g. of starch, or 234 g. of cane-sugar, or 256 g. of grape-sugar.

Exchange of food-stuffs. — The *transposal of the albumin* depends upon the amount of nourishment taken, and as well, indeed, upon the number of its calories and upon the amount of albumin taken in the food. If less albumin is administered, *where there is a sufficient number of calories*, than is contained in the albumin needed for nutrition, then more nitrogen is excreted in the urine than is contained in the food (1 g. of N corresponds to 6.25 g. of albumin). Should albumin be contained in the food given to cases where the total number of calories is sufficient, then an *equilibrium is formed* in the *nitrogenous production*, that is, the excreted N is equal to the amount of N administered. Should more albumin than is necessary be administered, in cases where the total number of calories is sufficient, then more N is excreted, and the equilibrium as far as N is concerned is soon reestablished.

Considerable proteid increase can be attained only in a person in the process of growth, in cases of convalescence from an acute disease, and in simple inanition.

If the total number of calories contained in the food is not sufficient, an increase of the excretion of nitrogen may take place even in cases where sufficient albumin has been taken as food.

Again, the albuminous exchange depends upon the previous nutrition and the condition of the body produced by that nutrition. Muscular people having plenty of albumin consume more albumin than fat people in whom the exchange of albumin is less.

Work has no influence on the consumption of albumin in general; but it favors the consumption of fats and carbohydrates. Should not sufficient fats and carbohydrates be

administered in food, the body will then consume its own fat, in order that the necessary work may be done.

However, everything in such cases depends upon the total number of calories; if it be very large, the contribution to the amount needed for work, where there has been no fat or carbohydrates administered, is made good in part by the metabolism of the albumin itself into the fat groups; and if the number of calories be insufficient, then both the fat as well as the albumin of the body are consumed.

ANOMALIES OF METABOLISM

The anomalies of metabolism thus far known consist of:—

1. *Qualitative changes* by which the urine will contain substances which are not excreted in it in health.

The most important qualitative changes occur in *diabetes mellitus*, where grape-sugar appears in the urine, while in health all the carbohydrates are transformed in the organism into CO_2 .

Certain forms of obesity seemingly depend upon the loss of the power to oxidize the fat formed in the body.

In some rare forms of disordered metabolism, which are not yet sufficiently known, some extremely peculiar substances are excreted in the urine; for example, *cystin* and *diamine* in cases of cysturia.

2. *Quantitative changes.* These are chiefly shown in the metabolism of the albuminoids. The laws of the equilibrium of N where the income is sufficient, which were mentioned in the previous paragraphs, suffer in a few diseases an alteration in the shape of a more extensive transformation: an increased consumption of albumin, a more marked oxidation of the albuminoids of the body takes place. This is noticed in fever, in many cases of phthisis, in carcinoma, in anæmia, and in leucæmia.

A diminution of proteid metabolism occurs in cases of convalescence from acute disease, in inanition, in many forms of obesity, and in myxœdema.

The conditions of diminished excretions as a result of disease of excreting organs, as, for example, the diminution of the excretion of urea in *nephritis*, belong to the disturbances of metabolism in the broader sense. The diseases of the pancreas, liver, and intestines ought to be here mentioned; in these conditions less fat and albumin are absorbed in the intestine than under normal conditions of the organs.

In gout it would appear that the question is one of an increased production of uric acid from an increased destruction of nuclein (see p. 206); the excess of uric acid in the blood can be partly accounted for by inflamed and necrotic areas of tissue (gout necrosis).

In order to diagnose a disturbance of metabolism with certainty, it is necessary to tabulate the income and the expenditure as in a balance-sheet. Clinically, the determination of the following data is sufficient:—

1. *The value of the food.*
2. *The constituents of the urine* (N, sometimes uric acid, finally sugar).
3. *The unabsorbed portion of food* in the fæces, determined by the amount of N and fat contained therein.

By determining these points the metabolism of the albuminoids can be accurately accounted for; the metabolism of the fats and carbohydrates will escape a quantitative estimation if the amount of carbonic acid in the breath be not measured.

1. The Value of the Food

In order to accurately determine this, it is necessary that everything which the patient eats should be weighed by the scales and what is left uneaten should be deducted. The value of the different articles of food in nutritive materials is cleared up by the following table:—

ARTICLES OF FOOD	ALBU- MIN PER CENT.	N PER CENT.	FAT PER CENT.	CARBO- HY- DRATES PER CENT.	ANALYSIS MADE BY
Raw beef, free from visible fat	18.36	3.4	0.9	—	Voit.
Moderately fat	20.91	3.3	5.19	0.48	König.
Fat, uncooked } Beef .	17.19	2.8	26.38	—	König.
Roast	30.56	4.89	6.78	—	Rubner.
Boiled	21.8	3.5	4.52	—	Renk.
Roast } Veal	18.88	3.02	7.41	0.071	König.
Raw	15.3	2.84	5.2	—	Renk.
One egg (45 g. without shell)	6.25 g.	1 g. N	4.9 g.	—	Voit.
Good milk	4.13	0.64	3.9	4.2	Voit.
Milk (for children) (Charité)	3.88	0.62	3.1	4.5	The author.
Skimmed milk	3.25	0.52	1.1	4.1	The author.
Butter	0.5	0.08	87.0	0.5	König.
Cheese	32.2	4.75	26.6	2.97	Renk.
Bacon (Charité) . . .	—	—	94.7	—	The author.
White bread (rolls) . .	9.6	1.5	1.0	60.0	Renk.
Rye bread, fresh . . .	5.63	0.9	—	44.0	The author.
Bread (Charité). . . .	8.22	1.315	0.64	58.3	The author.
Boiled potatoes, with- out the skins	2.18	0.35	—	23.0	Rubner.
Vegetables (Charité) from 3 analyses . . .	3.45	0.55	4.2	20.3	The author.
Soup (Charité) from 3 analyses	1.7	0.272	1.8	8.3	The author.
Beer (light)	0.56	0.09	—	5.5	The author.
Wine	0.19	0.03	—	2.0	König.
Coffee (weak)	0.25	0.04	—	—	The author.

2. The Constituents of the Urine

It is of prime importance to collect the urine passed in the 24 hours, without loss. The total amount of nitrogen contained therein is then determined according to the rules given on p. 206.

In *diabetes* a quantitative analysis of the sugar passed must be made (p. 196).

3. The Undigested Nitrogenous Materials and Fats left in the Fæces

The fæces passed on the day are marked by previously administering some of a black coloring charcoal mixture. The fæces are dried; the nitrogen in them is determined by the method of *Kjeldahl*, and the fat by extracting with ether.

It is customary to add the N in the fæces to the N in the urine and to record in the table under food the added figure as the expenditure.

The estimation of the amount in the fæces is both tedious and laborious; we can not do without it in some cases. For clinical purposes the values formed by Rubner as representing the consumption of food in the intestines may be used with advantage.

ARTICLE OF FOOD	N PER CENT.	FAT PER CENT.	CARBOHYDRATES PER CENT.
Meat	2.65	19.2	—
Eggs	2.9	5.0	—
Milk	8.9	5.7	—
Wheat bread	20.7	—	1.1
Rye bread	32.0	—	19.9
Potatoes	32.2	—	7.6
Vegetables	18.5	6.1	15.4

These values may only be used when a good-sized movement from the bowels occurs regularly; in many diseases associated with diarrhoea this consumption suffers to a great extent. That of fat is much affected in icterus and atrophy of the pancreas, in severe anæmias, and in most diarrhoeal diseases.

From the values obtained a **metabolic balance-sheet** is made something like the following:—

DISEASE: CARCINOMA OF THE STOMACH (AGE, 49 YEARS)

INCOME

DATE	BODY WEIGHT, POUNDS	NOURISHMENT	N	FAT	CARBO-HYDRATES	CALORIES
I. 12	115	1500 g. milk 85 g. bread 40 g. butter 4 eggs	7.8 1.1 — 4.0	16.5 0.54 34.8 19.6	61.5 49.5 — —	— — — —
Total			12.9	71.4	111.0	1474
I. 13	115	2000 g. milk 110 g. bread 40 g. butter 4 eggs	12.4 1.4 — 4.0	22.0 0.7 34.8 19.6	82.0 64.0 — —	— — — —
Total			17.8	77.1	146.0	1763
Average			15.35	—	—	1618.5

EXPENDITURE

DATE	URINE			FÆCES		N	TOTAL N
	Amount	Sp. Gr.	N	Moist	Dry		
I. 12	1350	1022	21.6	} 317	87.2	2.66	22.6
I. 18	1750	1015	23.4				24.4
Total							47.0
Average							23.5

Therefore the daily average is:—

Nitrogen income = 15.35

Nitrogen expenditure = 23.5

And the daily expenditure of 8.2 N = 241.1 g. of muscle tissue.

In this balance-sheet it is often necessary to estimate the N in reference to urea, or to albumin, or to muscle tissue. To render this estimate easy, the constant relations existing between them are here mentioned:—

Nitrogen :	Urea	:: 1 :	2.143
Nitrogen :	Albumin	:: 1 :	6.25
Nitrogen :	Muscle tissue	:: 1 :	29.4
Urea :	Nitrogen	:: 1 :	0.466
Urea :	Albumin	:: 1 :	2.9
Urea :	Muscle tissue	:: 1 :	13.71

The differential diagnosis of proteid metabolism is only called for in some special rare instances, as, for instance, in determining the malignant or benign nature of tumors.

The significance of these metabolic balance-sheets lies chiefly in the possibility of controlling by their study the *nutrition of the patient to the most exact degree*, and of always harmonizing the diet with each state of nutrition and body change.

In *diabetes mellitus* the constant attention paid to metabolism is of immediate significance in diagnosis and in treatment. Two varieties of this form of diabetes are recognized, either of which may become converted into the other:—

1. *The mild form*, in which sugar only appears in the urine after the *ingestion of carbohydrates*; the sugar excretion is larger or smaller according as the amount of carbohydrates ingested is greater or less.
2. *The severe form*, in which sugar is contained in the urine even after many days have passed in which *no carbohydrates* at all have been taken as food.

Only a carefully prepared metabolic balance-sheet will give an accurate knowledge of diabetics and their treatment.

As an example, I give a balance-sheet of a mild case of *diabetes*.

INCOME

DATE	BODY WEIGHT IN POUNDS	NOURISHMENT	N	FAT	CARBO- HYDRATES	CALORIES
III.	115	1 litre milk	6.2	31.0	45.0	—
15	—	10 eggs	10.5	49.0	—	—
	—	120 g. butter	—	104.4	—	—
	—	125 g. meat	4.2	1.1	—	—
	—	60 g. bread	0.8	0.4	35.0	—
Total			21.2	185.9	80.0	2600

EXPENDITURE

DATE	URINE				FÆCES		N	TOTAL N
	Amount	Sp. Gr.	N	Sugar	Moist	Dry		
III.	2800	1022	18.8	33.6 (1.2 %)	238	47.6	1.7	20.5
15								

Therefore of 80 g. of carbohydrates, 46.4 g. are consumed regularly, and 33.6 g. are excreted in the urine unconsumed. The excretion of nitrogen is somewhat less than the amount taken in.

SYMPTOMS OF A FEW OF THE METABOLIC DISORDERS

Diabetes mellitus. — The diagnosis is settled by a positive reaction to the test for sugar. It depends whether an examination of the urine is thought of at the right time. The following signs should induce the physician to make a urinary examination for sugar: diminution of physical and mental capacity, progressive cachexia, very much increased appetite (polyphagia), much increased thirst (polydipsia), large excretion of urine (polyuria); in certain cases there

is a tendency to the production of boils (furunculosis), simple wounds heal with difficulty, severe pruritus is present, the presence of certain diseases of the eye (cataract, optic neuritis), and an early development of impotence occurs. Concerning the mild and severe forms, see p. 238: ferric chloride reaction, p. 200.

Gout is characterized by repeated short attacks of inflammation of the joints, especially of the metatarso-phalangeal joint of the large toe, although it also occurs in other joints. After the attacks deposits of uric acid often accumulate in the cartilages of the joint (gout nodules, tophi). Tophi also are deposited in the cartilage of the ear and in the skin, especially of the leg. After frequent attacks deformities of the joints ensue. Interstitial nephritis and atrophic kidney (gout of the kidneys) often develop. In the intervals between the attacks there are many nervous disturbances (intervallary symptoms) and often inflammatory affections of the internal organs (visceral gout).

DISEASES OF THE THYREOID GLAND

Basedow's disease is characterized by struma, exophthalmus, tachycardia (often associated with dilatation of the left ventricle and a systolic murmur), tremor of the fingers, and nervous irritability. There is often cachexia.

Myxœdema (cachexia strumipriva) is characterized by absence of the thyreoid gland, a swelling of the skin of the entire body, gradual failure of the physical and psychological powers, loss of the hair, and progressive cachexia.

CHAPTER XI

DIAGNOSIS OF THE DISEASES OF THE BLOOD

As far as the history is concerned an inquiry into the hygienic conditions of the patient, his habits, and his occupation is of value, because psychical excitement, troubles, and worry often lead to anæmia. All conditions which lead to chronic *loss of blood* may be regarded as direct causes of anæmia: ulcer of the stomach and of the intestine, uterine myomata, profuse menstruation, certain intestinal parasites (the *Anchylostomum duodenale* and *Bothriocephalus latus*); finally, anæmia is produced by all those severe *disturbances of digestion* which are occasioned by atrophy of the mucous membrane of the stomach and of the intestines, chronic intestinal catarrh, and continued attacks of diarrhœa. Every severe injury to the organism, as well as every disease of long standing (syphilis, for example), may lead to true anæmia. However, many blood diseases develop without a discoverable etiological cause; in such cases the history should concern itself in part in inquiring carefully about the indefinite symptoms which usually precede the full development of the disorder, such as malaise, loss of energy, disturbed sleep, headache, cardiac palpitation, dyspepsia often, etc.

A diagnosis is directed to a disease of the blood when there is an intense *pallor* of the skin and the mucous membranes (see p. 7), associated with physical weakness.

As has been already mentioned among general symptoms, a blood disease may be *secondary*, that is, caused by a severe visceral disease which leads to bodily wasting, such as tuberculosis, carcinoma, amyloid degeneration, etc. Only when such a disease can be excluded may a diagnosis of a specific disease of the blood be considered; *an examination of the blood* will then substantiate it.

It is often possible to diagnose an essential (not a secondary) disease of the blood from the peculiar color of the skin. The skin

in pernicious anæmia is waxy yellow, often having a greenish tinge; this color is wholly characteristic.

The examination of the blood should concern itself with —

1. The macroscopic appearance of the blood.
2. The ordinary microscopic examination.
3. The counting of the corpuscles.
4. The measuring of the corpuscles.
5. The preparation of *stained* blood specimens.
6. The determination of the amount of hæmoglobin.

The scientific analysis of blood diseases includes in addition, among other things, the *reaction*, the amount of *carbonic acid* it contains, and the investigation of its *metabolism* (see previous chapter). A *spectroscopic* examination is necessary for diagnostic purposes in many cases of poisoning.

For the purposes of examination blood should be obtained by pricking the finger tip or the lobe of the ear, which has been previously washed and dried. The prick should be made with a sharp needle or, better, with a vaccination lancet; it should be deep enough that the blood will exude of its own accord; pressure must not be used; the first drop is wiped away, the second alone is to be examined.

1. The Macroscopic Examination of the Blood

includes the *color*, which normally should be of a bright red, and which in disease becomes paler and approaches a white. The rapidity with which it coagulates is also to be observed. If we accustom ourselves to prick as uniformly as possible, then the abundant or scanty amount exuding from the prick will furnish some conclusion as to the amount of blood. This can hardly be considered, though, in a differential diagnosis.

2. The Examination of the Fresh Blood Drop under the Microscope

A drop of blood is put on a slide, the cover glass carefully added; it is best to avoid evaporation and consequent drying by covering the edges of the cover glass with some warm paraffine. In the examination the following features should be observed:—

a. *The shape of the red blood corpuscles*; normally they have the shape of a disc with a central depression. The shape is unchanged in chlorosis and anæmia. In all severe anæmias changed forms appear, such as: *poikilocytes* (Fig. 40), which are club-shaped, pear-shaped, biscuit-shaped, or kidney-shaped red blood corpuscles; *microcytes*, red blood corpuscles much smaller than the ordinary red corpuscle; and *macrocytes*, which are decidedly larger than these.

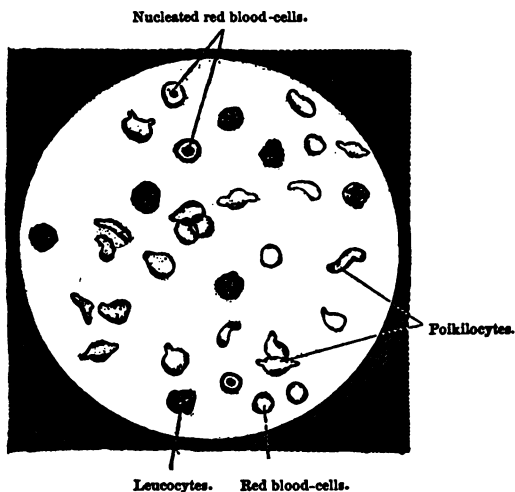


FIG. 40.—THE BLOOD IN PERNICIOUS ANÆMIA.

b. *The tendency to the rouleaux formation*; this tendency is absent in all conditions of extensive diminution in the number of red corpuscles, therefore in all severe anæmias.

c. *The number of the red blood corpuscles*; although this can only be positively determined by a counting apparatus, yet after practice in preparing specimens uniformly we can reach the position to tell whether the number is essentially diminished by the ordinary microscopic appearance. The increase of erythrocytes is the sign of anæmia.

d. *The color of the red blood corpuscles*, normally yellowish-red, is more or less pale in many diseases, especially in chlorosis.

e. *The number of the white blood corpuscles* and their relation to the red. Normally, 1 white exists to every 300 red blood corpuscles, or, in other words, in a microscopic field with open diaphragm and a high objective (Leitz 7), 3 to 5 white corpuscles will be present.

The abundant presence of leucocytes in a visual field (over 10) is an important sign of disease. Moderate increase of the leucocytes (1 white to 100 red) is called a *hyperleucocytosis* (see below). Very great increase of the leucocytes is the sign of *leucæmia* (1 to 50 and up to 1 to 2). These results should be controlled by the counting apparatus.

With practice with the ordinary microscopical examination we may be able to recognize, for instance, the nucleated red blood corpuscles as well as the variations in the white corpuscles. Yet these features are more easily recognized in examining stained preparations.

3. Counting the Blood Corpuscles

To count, the *Thoma-Zeiss* counting apparatus is used. It consists of a glass capillary tube that is blown out near the upper extremity into a bulb, which serves to draw up and to dilute the blood, and of a counting chamber. The blood is drawn up into the graduated tube to the mark 0.5 (or to 1), the point of the tube is carefully wiped, and then some of a 3 per cent. solution of sodium chloride (salt) is drawn into the tube until the whole contents of the tube reach the mark 101. The fluid in the tube is then thoroughly mixed by shaking (this is facilitated by the little glass ball which is found in the bulb). After this the mixture is placed in the counting chamber, which is exactly 0.1 mm. deep and the bottom of which is divided into microscopical squares. The space over each square is exactly $\frac{1}{1000}$ c.c. When placing the cover glass over the counting chamber, care should be taken to prevent the formation of air-bubbles. A large number of these squares should be counted, every 16 of which are marked off by darker lines, and in this way we obtain the average number of blood corpuscles

lying in each square. This average is then multiplied by 800,000 (if the blood was sucked up to the mark 1, it is only multiplied by 400,000), for the reason that the blood was diluted 100 times and the space over each square is $\frac{1}{100}$ c.c.; the result will give the number of red blood corpuscles in every cubic millimetre of blood.

The counting of the white corpuscles is performed in exactly the same way, although the blood is diluted in a separate pipette only 10 times. It is of advantage to add some methyl-violet to the diluting fluid, for the leucocytes take up this stain and appear much more prominently than otherwise. The destruction of the red blood corpuscles (by diluting the blood with 1 per cent. of acetic acid) is recommended in counting the white corpuscles.

In healthy men the number of the red blood corpuscles is 5,000,000, in women between 4,000,000 and 5,000,000, in every cubic millimetre. In *chlorosis* the number is not at all or but little changed, but in all anæmias it is much diminished, even down to 500,000; in severe leucæmia there is also a diminution of the number of red blood corpuscles.

The number of white blood corpuscles in health is 5000 to 8000 in every cubic millimetre. An increase in the number (*hyperleucocytosis*) occurs physiologically during the digestion of the *albuminoids* (10,000 to 20,000) and occurs pathologically in many infectious and cachectic diseases, *e.g.*, pneumonia and carcinoma (see p. 250). Only an increase to over 50,000 in a cubic millimetre would justify a diagnosis of *leucæmia*; this diagnosis will be probable if with an existing hyperleucocytosis the number of leucocytes increases in a short time.

4. Measuring the Blood Corpuscles

The size of the red blood corpuscles may be well estimated and macrocytes and microcytes recognized with sufficient distinctness. For a careful examination a *micrometer* eyepiece should be used. The red blood corpuscles of the healthy vary in size from 6.5μ (microns), on the average they are 7.6μ ; they are of equal size in the same individual. Macrocytes are those whose size is 10 to 12μ , gigantocytes 12 to 15μ . Their presence denotes a grave anæmia. The size of the leucocytes varies extremely.

5. The Preparation and Microscopical Appearance of Stained Specimens

(According to *Ehrlich*)

The drop of blood is taken up from the finger tip directly on to the cleansed cover glass, which is placed lightly on another clean cover glass in such a manner that the edges do not coincide and then the glasses are drawn apart without exerting pressure, which must be avoided. Care should be taken that the fingers do not touch the cover glasses, because the warmth and moisture of the skin will change the very sensitive blood corpuscles. The blood has now been finely spread in a thin layer on both cover glasses. The spreads are now dried by the air and thereafter fixed by heat. The heating must be gradual, for which purpose the cover glasses are placed in a hot-air oven or on a copper plate; the latter is heated at one end to 120°C. and kept at this temperature for 2 hours. After they have cooled they are ready for staining.

The dye most frequently used is the eosin-hæmatoxylin solution (hæmatoxylin, 2; alcohol, glycerine, and distilled water, aa 100; glacial acetic acid, 10, to which alum in excess is added; the solution should stand several weeks, then a small quantity of eosin is added to it). The spreads are put in this solution and kept there for 30 minutes, when they are removed and washed with water; the red blood corpuscles will appear red, the nuclei of the white and those of the red are deeply stained bluish-black, and the eosinophile granulations (see below), red; the protoplasm of the white blood cells is almost unstained, having only a pale-red tint.

Ehrlich's three-color mixture (see p. 144) is very serviceable for staining the blood. This stains the nuclei a greenish-blue, the eosinophile granulations red, the red blood cells orange.

Beautiful results are obtained with the eosin-nigrosin-aurantia-glycerine mixture. (To 1 volume of glycerine saturated with aurantia, add 1 to 2 volumes of glycerine; shake the mixture thoroughly and then add eosin and nigrosin in excess; saturation results after long-continued shaking.) The hæmoglobin takes up the yellowish-red tint of the aurantia, all nuclei are gray or black, and the eosinophile granules are red.

The stained preparations are best examined with an oil immersion lens and with open diaphragm.

In a stained specimen we recognize:—

1. The nucleated red blood corpuscles; these are always a sign of severe blood disease; they are present in many anæmias, and occur less seldom in leucæmia; nucleated megalocytes and gigantocytes indicate very grave anæmia. Still in spite of their presence, improvement, even a cure, may result.

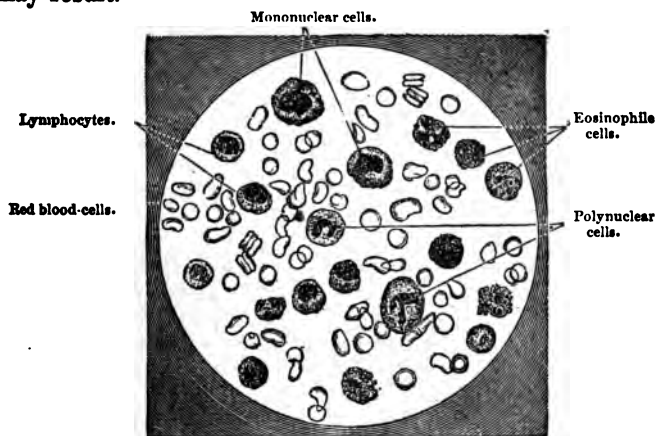


FIG. 41.—SCHEMATIC REPRESENTATION OF VARIOUS KINDS OF LEUCOCYTES.

2. The different forms of the *white* blood corpuscles:—

- a. *Lymphocytes*, of varying size; usually somewhat larger than a red blood corpuscle, possessing a round nucleus and small protoplasmic body; they arise from the lymphatic glands, and a marked increase of them indicates *lymphatic leucæmia*.
- b. *Mononuclear leucocytes* are much larger than the red corpuscles, and have a large ovoid nucleus and a large protoplasmic body; from them are developed the
- c. *Polynuclear leucocytes*, which have a polymorphous nucleus, and form the mass of the leucocytes;

they are chiefly present in pus. Mono- and polynuclear cells are increased in *splenic* and in *myelogenic leucæmia*.

- d. *Eosinophile cells*, large, round, and nucleated; they are characterized by the glistening granules in the protoplasmic body of the cell; these granules are deeply stained by eosin. These cells arise in the bone-marrow, are seldom found in normal blood, and when abundantly present justify the conclusion that there is an implication of the marrow of the bone.

6. The Determination of the Amount of Hæmoglobin in the Blood

The amount of hæmoglobin in the blood is determined with sufficient accuracy by means of *Fleischl's hæmometer*. The color of the blood diluted with water is compared with a wedge of glass which is colored purplish-red. The blood is sucked up in a capillary tube of known dimensions, dissolved in water in the one compartment of a glass box which is partitioned into two divisions; under the other compartment, which is filled with water alone, the colored glass wedge, whose color increases in intensity as the wedge increases in depth, moves across. The wedge is provided with a scale, empirically determined in such a manner that the 100 mark of its color corresponds to the normal hæmoglobin in the blood. Under the glass bottom of the box a light is reflected by a gypsum plate; should then the color be alike in both compartments, the number indicated on the scale at this stage will denote the hæmoglobin contents of the blood. The limits of error in this instrument are within 15 per cent. *Gower's hæmoglobinometer* determines the amount of the coloring matter of the blood by comparing a specimen of diluted blood with some artificially colored glass rods. Pretty accurate results are obtained by the expert who uses photometric spectral analysis suggested by *Vierordt*; but its use is extremely difficult.

The amount of hæmoglobin in the blood is much diminished in chlorosis, while the number of red corpuscles is

not at all affected. In the other anæmias a diminution in the amount of hæmoglobin will coincide with a decrease in the number of red blood corpuscles.

The absolute amount of hæmoglobin is 13 to 15 g. to every 100 c.c. of blood, in women usually a little less than in men.—Hæmoglobin is converted into *albumin* and *hæmatin*. Hæmatin with hydrochloric acid produces *hæmin*, which form in beautiful crystals (*Teichmann's*), by whose presence the slightest trace of blood may be recognized.

Teichmann's blood test.—A small quantity of dried blood, to which 1 to 2 drops of glacial acetic acid and a small crystal of common salt are added, is heated in a glass dish over a full flame until it boils; it is then permitted slowly to evaporate, when numerous brownish-yellow needles and crystals of hæmin will be formed.

The reaction of the blood is alkaline; the alkalinity is diminished in severe anæmias, in fever, in severe diabetes, and in the emaciation stage of carcinoma.

The reaction of the blood may not be determined simply with litmus, etc., on account of the individual color of the blood and chiefly because the blood contains different acids and bases in varying degrees of saturation. In one sense the alkalinity of the blood may be reckoned from the amount of *carbonic acid* the blood contains, for there is an approximately standard relation between the alkalescence and the amount of CO_2 .

The determination of the *specific gravity* has thus far been of little use for practical diagnostic purposes; in health it varies between 1045 and 1075.

The *spectroscopic* examination of the blood is of importance in diagnosing carbonic oxide intoxication.

Normal blood strongly diluted with water will show the absorption bands of *oxyhæmoglobin* in the yellow and green (between the Fraunhofer lines of D and E). On adding a few drops of a solution of ammonium sulphide, the two lines will vanish and a single line (between D and E) of reduced hæmoglobin will appear.

The bright red blood of carbonic oxide intoxication shows likewise, when viewed in the spectroscope, two lines between D and E,

though these appear closer to each other than the oxyhæmoglobin lines. On the addition of ammonium sulphide the lines of the CO hæmoglobin do not disappear.

In cases of poisoning with *potassium chlorate*, anilin, antifebrin, phenacetin, etc., the color of the blood is like chocolate, and in the spectroscope, in addition to the two oxyhæmoglobin lines, an absorption line is observed in the red which belongs to *methæmoglobin*. On adding some ammonium sulphide, all three lines disappear and the one line of reduced hæmoglobin puts in an appearance.

CHIEF SYMPTOMS OF THE MOST IMPORTANT DISEASES OF THE BLOOD

Chlorosis occurs in young persons, especially in young girls; still sometimes it occurs in women, especially after parturition. It is characterized by pallor of the skin, intense fatigue, often by dyspepsia, palpitation, etc. The essential changes in the blood are a *great diminution* in the *amount of hæmoglobin* without an essential decrease in the number of red or an increase in the number of white blood cells. The prognosis is usually good.

Hyperleucocytosis is a temporary increase in the number of white blood corpuscles; it is a symptom of many inflammatory diseases (occurs principally in pneumonia, erysipelas, meningitis, and in the cachectic diseases, especially in *carcinoma*). In typhoid fever, malaria, glanders, and in many forms of sepsis hyperleucocytosis is absent. It occurs physiologically during digestion. Hyperleucocytosis may extend to an increase to 100,000 leucocytes in one cubic millimetre of blood. The characteristic differential point between it and *leucæmia* lies in the demonstration of the original disease and in the increase of the polynuclear leucocytes alone. The prognosis is dependent on the original disease producing it.

Leucæmia is characterized by a great increase in the number of white blood corpuscles, so that the relation may be

from 1 to 60, to 1 to 2. It is to be differentiated from the primary stage of a leucocytosis in the more rapid increase of the leucocytes in leucæmia. The red cells are usually diminished in number, are often nucleated, and the amount of hæmoglobin is also diminished. The following forms exist, although *each may merge into the others*.

1. *Lymphatic leucæmia*. Swelling of all the lymph glands; the lymphocytes of the blood are increased.

There is a well-characterized form of **acute leucæmia** in which only the lymphocytes are increased. It runs a rapidly fatal course, accompanied by the clinical phenomena of hæmorrhagic diathesis, dyspnœa, enlargement of the lymph glands and spleen; in many organs leucæmic hyperplasia appears. The uric acid excretion is enormously increased.

2. *Myelogenic leucæmia*. There are many eosinophile as well as mononuclear leucocytes and nucleated red blood corpuscles.

3. *Splenic leucæmia*. The spleen is very large. The blood contains many eosinophile and mononuclear cells. A progressive cachexia characterizes all three forms, which terminate fatally.

Pseudo-leucæmia is the name given to the disease which has the clinical features of leucæmia, such as cachexia, lymphatic gland enlargements, and a splenic enlargement, but which runs its course *without the changes in the blood characteristic of true leucæmia*. The number of leucocytes is normal and there is little diminution of the red cells and of hæmoglobin.

Pseudo-leucæmia, presenting large swellings of the lymph glands, is called *Hodgkin's disease*.

Pernicious anæmia. The number of red blood cells is much diminished, even to 400,000 in each cubic millimeter. Poikilocytes, macrocytes, and microcytes are in abundance. There are nucleated red corpuscles and nucleated giganto-

cytes. The amount of hæmoglobin is relatively increased, while the number of leucocytes is normal or even diminished. The prognosis is most grave.

Secondary anæmia occurs in the course of severe dyspepsia, anchylostomiasis, carcinoma, phthisis, tertiary syphilis, malaria, amyloid degeneration, chronic intoxications, etc. There is a large decrease of the red blood cells; macrocytes and microcytes are present, but rarely are there any nucleated red cells; the amount of hæmoglobin is also diminished. The number of polynuclear leucocytes is *increased*. The prognosis of secondary anæmia depends on its causative disease; should we be successful in removing that, the anæmia may be cured. Secondary anæmia may go into pernicious anæmia; on this account in some cases a differential diagnosis may be extremely difficult.

CHAPTER XII

ANIMAL AND VEGETABLE PARASITES

I. ANIMAL PARASITES

THE animal parasites which are found in or upon the human body are, in part, harmless organisms which infest the skin or the intestines and are without diagnostic significance; in part, however, they are productive of diseases, more or less severe, the treatment of which is dependent almost wholly upon a correct diagnosis.

In the following summary, the principal animal parasites are included:—

I. Protozoa.

- a. Rhizopods: Monads and *Amœba coli*.
- b. Sporozoa: Coccidia.
- c. Infusoria: *Cercomonas intestinalis*, *Trichomonas intestinalis*, *Paramecium coli*.

II. Vermes (Worms).

- a. *Tape-worms (Cestodia)*.
 - 1. *Tænia solium*.
 - 2. *Tænia mediocanellata* or *saginata*.
 - 3. *Bothriocephalus latus*.
 - 4. *Tænia nana*.
 - 5. *Tænia flavopunctata*.
 - 6. *Tænia cucumerina*.
 - 7. *Tænia echinococcus*.
- b. *Flukes (Trematoda)*.
 - 1. *Distoma hepaticum*.
 - 2. *Distoma lanceolatum*.
 - 3. *Distoma hæmatobium*.
- c. *Thread worms or Round worms (Nematoidea)*.
 - 1. *Ascaris lumbricoides*.

2. *Ascaris mystax*.
3. *Oxyuris vermicularis*.
4. *Anchylostoma duodenale*.
5. *Tricocephalus dispar*.
6. *Trichina spiralis*.
7. *Anguillula intestinalis*.
8. *Filaria sanguinis*.

III. Arthrozoa.

1. *Acarus scabiei*.
2. *Acarus folliculorum*.
3. *Pediculi*.
4. *Pulex irritans*.

The Protozoa are practically without diagnostic significance. They are round, granular organisms, about $1\ \mu$ in length, some of the infusoria being a little larger, provided with ciliæ or flagellæ. They are found in healthy fæces, in the discharges of chronic diarrhoea, and sometimes in the normal vaginal secretions. But the amœba coli is the causative factor of dysentery, and has therefore diagnostic significance.

Tape-worms

The tape-worms are principally intestinal parasites. As such they evoke a complex of dyspeptic, dysenteric, and nervous symptoms which are, in a measure, exceedingly painful and which disappear upon the expulsion of the parasite. The diagnosis of the presence of a tape-worm can be established only by the passage of segments (proglottides) of the worm.

Tape-worms consist of a *head* (scolex) and *joints* (proglottides). They reproduce by sexual alternation. The bisexual segments bud from the head, the impregnated eggs entering the stomach of some second animal, the *intermediate host*. Here the coverings of the eggs are digested and the embryo becomes free. It reaches the tissues of the intermediate host in the form of a *cysticercus*. If the cysticercus reaches the stomach of man with his food, a new tape-worm is developed.

Tænia solium (intermediate host, the pig) attains a size of from 2 to 3 M., its proglottides are from 9 to 10 mm. long and from 6 to 7 mm. broad. The proglottides nearest the head are short and

gradually increase in size as the tail is approached. The head has the size of a pin. Under the microscope may be seen four sucking discs, usually pigmented, and a proboscis or rostellum with from 25 to 30 hooklets of different sizes (Fig. 42). The segments have lateral sexual openings and a uterus with few branches. The eggs are oval, about 0.036 mm. long and 0.03 mm. broad, with a thick covering and radial striæ. In the interior of the ovule, the hooklets of the embryo are visible. The larvæ (*Cysticercus cellulosæ*) are the size of a pea and may be deposited in the organs of the body (mainly the skin, the muscles, the brain, the eye) by auto-infection, if they have entered the stomach.

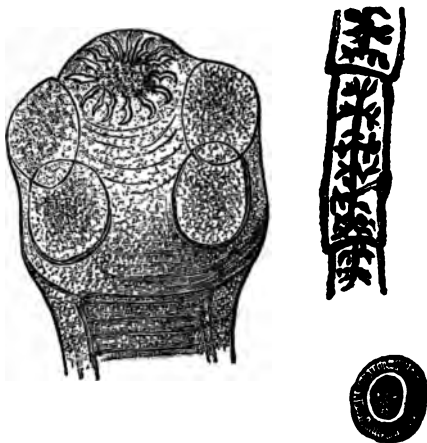


FIG. 42.—MICROSCOPIC PICTURE OF *Tænia solium* (HEAD, PROGLOTTIDES, Egg).

Tænia solium is the tape-worm most frequently found in the intestine. It is usually possible to distinguish this species with the naked eye or with a magnifying glass, by the delicacy and transparency of its segments and by the few ramifications (7 to 12) of its uterus.

The diagnosis of *cysticercus cellulosæ* of the skin is usually easy to make. Multiple movable tumors, from the size of a pea to that of a bean, are observed. Excision and examination makes the certainty of diagnosis more positive. The presence of a cysticercus in the eye may be demonstrated ophthalmoscopically. The diagnosis of cysticercus in the brain is made probable, if focal cerebral symptoms appear without demonstrable etiology, and if, at the same time, cysticerci are present in the skin or in the eye.

Tænia saginata or *mediocanellata* (intermediate host, cattle) are from 4 to 5 M. long. The head has no rostellum or circle of hook-

lets, but is provided with four very powerful sucking discs (Fig. 43). The proglottides are longer than those of *Tænia solium* and do not taper as they approach the head. The generative cloaca is situated



FIG. 43. — MICROSCOPIC PICTURE OF *TÆNIA SAGINATA* (HEAD, PROGLOTTIDES, EGG).

at the sides and the uterus has many ramifications. The eggs are somewhat more oval than those of *Tænia solium*, but otherwise the resemblance is marked, although the hooklets of the embryo are not visible. The larvæ does not develop in the tissues of the human body.

The segments of a *Tænia mediocanellata* may be recognized with the naked eye; they are thicker and not so delicate as those of *Tænia*

solium and the uterus has many more ramifications (from 15 to 20).

Bothriocephalus latus (intermediate host, various fishes, pike, salmon, etc.; geographical distribution limited mainly to the coast of the Baltic and Switzerland) is from 4 to 15 M. long. Its head is 2 mm. long, 1 mm. broad, club-shaped, and is provided in the median line with grooves which act as sucking discs (Fig. 44). The segments nearest the head are short and narrow, those further away almost quadrilateral. The uterus, when filled with eggs, is brown in color and presents a star-shaped form. The ovules are oval, 0.07 mm. long, 0.045 mm. broad, have a brownish shell, and are provided with a little cover.

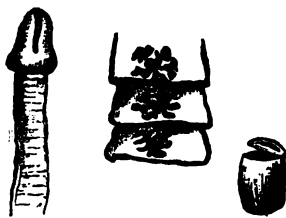


FIG. 44. — MICROSCOPIC PICTURE OF *BOTHRIOCEPHALUS LATUS* (HEAD, PROGLOTTIDES, EGG).

The diagnosis of *Bothriocephalus latus* is of importance, since its presence may evoke a profound *anæmia*, which disappears upon the expulsion of the tape-worm. The proglottides of *bothriocephalus* may be recognized by their brown color and by the rosette-shaped uterus.

Tænia nana is from 10 to 15 mm. long, 0.5 mm. broad. The diameter of the head, which has a rostellum and sucking discs, is 0.3 mm. The segments are short, 4 times as broad as they are long. The uterus is oblong in shape. The ovules measure from 0.03 to 0.04 mm. in diameter; they are surrounded by a double membrane without a striated shell. In the interior of the egg, the embryo with its hooklets is visible. From 4000 to 5000 of these tape-worms may infest the intestines simultaneously. *Tænia nana* has been seen only in southern countries (Italy, Egypt), and is said to call forth severe psychical and nervous disturbances.

Tænia flavopunctata and *Tænia cucumerina* are extraordinarily rare.

Tænia echinococcus is found in the human body only in its larval stage.

The tape-worm itself exists only in the intestines of dogs. It is 4 mm. long. The head possesses a circle of from 20 to 30 hooklets. The embryo reaches the human stomach and intestine and becomes encysted. The cyst-wall consists of two layers, an outer, finely lamellated layer, the cuticula, formed of chitinous material, and an inner layer, the parenchymatous, which contains muscle fibres and blood-vessels. The scolex or head develops in the parenchymatous layer and is provided with hooklets and sucking discs. The echinococcus cyst may be *unilocular* or secondary or *daughter cysts* may develop within it, or it may consist of a mass of small cavities, filled with a colloid fluid, the walls of which may show concentrically arranged layers. This is known as a *multilocular echinococcus cyst*.

Echinococcus cysts have their principal seat in the liver, but are found, less often, in the lungs, the brain, or the heart.

The symptoms evoked are those of a large cyst the nature of which is determined by aspiration and microscopic exam-

ination of the aspirated material. Sometimes, characteristic *membrane* and *hooks* (Fig. 45) are seen, or *chemical* examination of the fluid discloses some of its peculiar properties.

The fluid of an echinococcus cyst is usually clear, of a specific gravity of from 1008 to 1013. It contains *little* or *no* albumin, but *sodium chloride* is present in large quantities, and frequently grape-sugar and succinic acid are found.

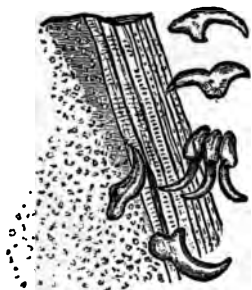


FIG. 45. — ECHINOCOCCUS MEMBRANE AND HOOKLETS.

The presence of succinic acid may be determined as follows: the fluid is heated over a water bath, is made acid by the addition of hydrochloric acid, and is then shaken up with ether. The ether is evaporated and if succinic acid is present, a crystalline jelly remains, which is to be dissolved in water. With ferric chloride, succinic acid gives a rust-colored, colloid precipitate which on heating in a test-tube gives off a vapor exceedingly irritating to the mucous membranes.

Flukes (Trematoda)

Distoma hepaticum is of a leaf-like form, has a short, globular head, and attains a length of 28 mm. The ovules are oval, 0.13 mm. long, 0.08 mm. broad, and are provided with a cover (Fig. 46). This parasite is rarely found in the biliary passages in man, and its eggs have been occasionally found in the intestines. Its diagnostic importance is insignificant, but the possibility of its confusion with eggs of important diagnostic worth must be mentioned.



FIG. 46.
EGG OF *DISTOMA*
HEPATICUM.

Distoma hæmatobium occurs only in the tropics. Its residence is in the portal system of veins and

in the veins of the bladder and of the rectum. It causes diarrhœa, hæmaturia, and ulcerations of the mucous membranes.



FIG. 47.
EGG OF *DISTOMA*
HÆMATOBIUM.

The male worm has a length of from 12 to 24 mm., the female from 16 to 19 mm. The abdomen of the male has a sulcus opening downward, in which the female is carried. The eggs (Fig. 47)

are found in the lungs, the liver, and the bladder, are 0.12 mm. long and 0.04 mm. broad, and are provided at the end or side with a thorn-like projection.

Distoma lanceolatum is a lancet-shaped worm, 7 to 8 mm. long. 2 to 3.5 mm. broad, smaller than but similar to *Distoma hepaticum*. The parasite is very rarely found in the gall-bladder or gall-passages, and the eggs are still more rarely seen in the fæces. Its diagnostic importance is slight.

Thread Worms or Round Worms (Nematoidea)

Ascaris lumbricoides is the common thread worm. The male attains a length of 25 cm., the female of 50 cm. It appears in large numbers in the human small intestine. In general it is a harmless parasite, but sometimes reflex convulsions in children are attributed to it. The eggs are found in the fæces in large numbers. They are round, yellowish-brown, of a diameter of 0.06 mm. In the fresh state, they are surrounded by a crenated, albuminous covering (Fig. 48), which in turn surrounds a thick, concentric, striped shell which contains a granular material.



FIG. 48.

EGG OF ASCARIS
LUMBRICOIDES.

Oxyuris vermicularis, thread worm or pin worm.



FIG. 49.

EGG OF OXYURIS
VERMICULARIS.

The male is 4 mm., the female 10 mm. in length. The parasites are found in the intestines in large quantities. The eggs are 0.05 mm. long and 0.02 mm. broad, are oval and more pointed at one end than at the other. They have an edge with a double contour (Fig. 49). The worm often leaves the intestine, and, remaining in the neighborhood of the anus, evokes a most troublesome itching.

✓ *Anchylostoma duodenale* is diagnostically of the greatest importance, because, by its constant sucking of blood from the walls of the intestine, it produces a profound anæmia which may resemble, in its clinical course, a case of pernicious anæmia.

Anchylostoma is seen in brickmakers, miners, and workmen in tunnels, and when an anæmia presents itself in laborers of this class, the fæces must always be examined

for anchylostoma. As long as no anthelmintic is administered, only the *eggs* are found in the stools.

The male is from 8 to 12 mm., the female from 10 to 18 mm. long. The male has a three-lobed tail, the female a pointed, conical tail. The cephalic end is provided with a bell-shaped mouth-capsule which contains four claw-like teeth. The eggs (Fig. 50) are 0.05 mm. long and 0.03 mm. broad. They have a smooth surface and in the interior several *segmentation bodies* are visible. If the ovules are not absolutely recognizable at first, the fæces may be allowed to stand in a warm



FIG. 50.

EGG OF ANCHYLOSTOMA
DUODENALE.

place for 2 or 3 days, when microscopic examination will disclose in anchylostoma eggs a decided increase in the segmenting process; or the patient may be given an anthelmintic, such as the extract of *felix mas*, in order to establish the diagnosis by the appearance in the fæces of the parasite itself.

Trichocephalus dispar (Fig. 51) is found in the large intestine, and is not of great diagnostic importance. The male is 4 cm., the female 5 cm. long. The eggs, 0.06 mm. long and 0.02 mm. broad, appear in large numbers in the stools, are brown in color, and are closed at each pole by a shining cover.



FIG. 51.

EGG OF TRICHOCEPHALUS
DISPAR.

Trichina spiralis is found in the human body in the *muscles* and in the *intestines*. By the eating of improperly cooked, trichinous pork, the trichinæ reach the human stomach and intestine. Here the capsule containing them is dissolved and males (1.3 mm. long) and females (3 mm. long) become free, and multiply. In the course of from 5 to 7 days, the young trichinæ work their way through the intestinal wall, enter the blood-current, and are carried into the muscles, where they may become encapsulated (Fig. 52). The diagnosis of trichinæ is established by the appearance of the parasite in the fæces after the administration of anthelmintics — although this is rarely accomplished — or by the finding of trichinæ in the muscles. The

symptoms of inspection by trichinæ depend upon the stage of the disease. If the parasites are still in the intestines, the symptoms of a gastro-enteritis present themselves; if they have reached the muscles, multiple abscesses in these organs appear.

Anguillula intestinalis (*Rhabdonema strongyloides*, *Leuckart*) are 2.25 mm. long, with rounded, obliquely striped bodies, and appear in large numbers in the small intestine. The eggs bear a striking resemblance to those of *anchylostoma duodenale*, and a differentiation between the eggs of these species may, occasionally, be of importance. They are not known to exert any injurious influence.



FIG. 52.

TRICHINÆ IN MUSCLE.

Filaria sanguinis occurs chiefly in the tropics, evoking hæmaturia and chyluria. Great numbers of the embryos circulate in the blood. The parasite is an auto-mobile, delicate worm, surrounded by a thin membrane. It is 0.35 mm. long and about as broad as a red blood-cell. In the sediment of the urine the embryos may be found in abundance.

Filaria medinensis, also a tropical parasite, is a very long (80 cm.), very narrow worm (about 1 mm. broad). It produces a severe furunculosis.

Arthrozoa

The *head-louse* (*Pediculus capitis*), the *body-louse* (*Pediculus vestimenti* or *corposis humani*), and the *crab-louse* (*Pediculus pubis*) must be given diagnostic consideration, since through their bites *eczema* and *excoriations* may be evoked, which may be confused with other skin diseases, and hence be wrongly treated.

The *flea* (*Pulex irritans*) and the *bed-bug* (*Acanthia lecticularis*) must be mentioned in this connection, since the flea-bite bears some resemblance to *patechia*, and may occasionally mislead to a diagnosis of *purpura*. The wheals arising from bed-bug bites look something like *roseola*.

Acarus scabiei, the itch-mite, is the etiological factor of the itch or scabies, which is recognized by the characteristic cuniculi made in the skin by the female, and the accom-

panying eczema. The male itch-mite is 0.2 mm. long, 0.35 mm. broad. The female is 0.35 mm. long, 0.5 mm. in width. Examined microscopically, the itch-mite bears some resemblance to a turtle with a conical proboscis and eight legs.

Acarus folliculorum, the pimple-mite, is found among the contents of hair-follicles (comedones). It is of worm-like form, 0.02 mm. broad, 0.1 mm. in length.

II. VEGETABLE PARASITES

1. Moulds and Fungi

The moulds are flowerless plants (cryptogamous) without stems or leaves, with simple foliage (thallophytes). The foliage consists of simple cells without nuclei and devoid of chlorophyll. They never multiply by fission but by the formation of long chains of cells (*hyphæ*). By the interbranching of these chains, a dense basket-work may arise (*mycelium*). Single hyphæ show peculiarities of growth, the *fruits* of the plants developing upon them. These fruits are called *spores* or *conidia*. According to the manner in which the fruit-bearing hyphæ develop from the mycelium and the manner in which these in turn form the conidia, the schizomycetes are divided into different groups: *mucorinæ*, *aspergillæ*, *penicillia*, etc.

The *saccharomycetes* do not form hyphæ nor mycelia; they consist simply of single cells without nuclei or chlorophyll. They multiply by *gemination*. A bud appears on the surface of the mother-cell, which increases in size and finally detaches itself. Frequently, large masses of these cells cling together and form *colonies*.

There are *intermediate forms* between the moulds and fungi, which, under some conditions of nutrition, form hyphæ, and under other circumstances grow in colonies. The principal member of this group is the *Saccharomyces* or *Oidium albicans* (Soor fungus).

Achorion Schönleini, the parasite of *favus*, was the first of the vegetable parasites of man to be recognized.

Trichophyton tonsurans is the fungus of *herpes tonsuraus* and *parasitic sycosis*.

Both of these fungi have mycelia with many branches, with distinctly jointed hyphæ. In the fungus of favus the branches usually stand at right angles.

These fungi may be raised in characteristic pure cultures. A typical favus or herpes may be brought about by inoculation in the skin.

Microsporon furfur is the parasite of *Pityriasis versicolor*. The proof of the presence of this fungus is of diagnostic importance, since the yellowish scales of pityriasis — mostly seen in the cachectic diseases, particularly phthisis — may be easily taken for an actual pigmentation of the skin. The scales of pityriasis are easily removed and show under the microscope, particularly well upon the addition of a few drops of caustic potash, an entangled mycelium with heaps of shining conidia.

Aspergillus and *mucorina* are occasionally found in the external auditory passages, in the nostrils, and in the nasopharynx. The diseases they call forth, however, are evoked simply in a mechanical manner by their presence. The growth of fungi in the lungs (pneumonomycosis aspergillina) is usually a secondary deposit in necrotic tissue or cavities already existing.

Saccharomycetes often appear in the fermenting contents of the stomach, mainly in cases of dilatation, chronic catarrh, and carcinoma. By the action of this fungus sugar is split up into alcohol and carbonic acid gas.

Thrush fungus (*Saccharomyces* or *Oidium albicans*) has the power of producing necrosis of pavement epithelium, and is the cause of the grayish-white membranous deposits on the mucous membrane of the mouths of poorly nourished children and profoundly sick people. These membranes may arise in other organs of the body provided with pavement epithelium, as the œsophagus and vagina. In media rich in sugar and acid in reaction, this parasite grows in pure culture, as in the stomach; in alkaline media, as in the mouth, it grows with abundant hyphæ and conidia.

2. Schizomycetes (Bacteria)

In the category of these minute beings (micro-organisms) belong the producers of the infectious diseases. In the cases of some of the infectious diseases, the clinical demonstration of the specific micro-organism is indispensable.

Biology of Bacteria

The bacteria constitute the lowest plane of vegetable life. They appear in the following forms:—

1. *Micrococci* or *round bacteria* are arranged in chains (*streptococci*), or in grape-like bunches (*staphylococci*), or in pairs (*diplococci*).

2. *Bacilli* or *rod-shaped bacteria* may appear with curves (*vibriones* or *comma bacilli*), or they may grow into long chains (*leptothrix*).

3. *Spirilla*, screw form.

A dense mass of bacteria, bacilli, or cocci is known as a *zooglæa mass*.

Bacteria multiply by successive fission. Many bacteria, however, increase by *spores*. In the mother bacterium a granular, strongly refracting area becomes differentiated, is freed, and grows into a new bacterium. The spores represent the *permanent form* of the bacteria, which die very soon upon the application of a moderate degree of heat (50° to 60° C.) or under the influence of a somewhat concentrated antiseptic solution (3 per cent. solution of carbolic acid). The spores, however, are very resistant to every external influence, and can be killed with certainty only after being subjected for half an hour to the influence of boiling water, or for 3 hours to a dry heat of 110° C. The spores are not rendered innocuous with certainty by the usual dilutions of antiseptic solutions. *Pathogenic* and *non-pathogenic* bacteria are distinguished. The latter do not develop in the human body, but thrive on dead material (as *saprophytes*), producing *fermentation* and *putrefaction*.

Pathogenic bacteria thrive in the bodies of man and the lower animals, producing the *infectious diseases*; some of them, however, like the anthrax bacillus, can live on dead material. This variety is called *ectogenous* or *facultative*.

The Demonstration of Bacteria

For clinical purposes the main examinations of bacteria are confined to pus, sputum, aspirated fluids, fæces, and blood; for such study the mounting and staining of *dry specimens* is sufficiently satisfactory. In some cases, however, this method does not suffice, and the preparation of a pure culture (*Koch*) or animal inoculation must be resorted to.

The preparation of a dry specimen. A small particle of the substance to be examined is placed upon a perfectly clean *cover glass*, upon which another cover glass is then placed with great care. The two cover glasses are then repeatedly drawn over each other in order to get as fine a layer as possible on either glass. It is then allowed to dry as thoroughly as possible *in the air*, the prepared surface, of course, lying uppermost. The cover glass is then seized with a forceps and is passed two or three times horizontally through the flame of a Bunsen burner or an alcohol lamp. This is for the purpose of coagulating the albumin present. The specimen may now be subjected to the staining fluid.

If one wishes to make a hasty examination, the substance to be examined may be transferred at once to a *slide*, which, after heating in the flame, may be further treated the same as the cover glass.

The staining of a dry specimen. Alcoholic solutions of the *basic aniline dyes* must be kept at hand. The main ones are Bismarck brown, methylene blue, methyl-violet or gentian-violet, fuchsin (red), and malachite (green). To prepare these solutions, the crystalline powder of the dyes is dissolved in alcohol in excess, thoroughly shaken, allowed to stand several hours, and filtered. Four or five drops of the saturated alcoholic solution are placed in a watch-glass full of distilled water. The dry specimen is placed into this solution, the prepared side *down*, for from 2 to 4 minutes. It is then washed in water, dried between two pieces of filter paper, laid upon the slide in oil of cloves or Canada balsam, and is examined under the microscope. An *oil immersion* objective, an *open* diaphragm, and an *Abbé* condenser are essential for a proper bacteriological examination.

In order to stain rapidly, a concentrated watery solution may be dropped directly upon the specimen. Slide preparations are always treated in this manner, the staining fluid being washed off with water. They are microscopically examined without cover glasses.

The method just described answers ordinary clinical demands. The aniline dyes stain micro-organisms and the nuclei of the cells intensely; the protoplasm of the cell is usually stained very weakly.

By staining bacteria by *Gram's* method, they can be *isolated*, so far as coloring is concerned, from the tissues in which they lie. This method consists in placing the cover glass preparation in an aniline-gentian-violet solution for 3 minutes (see below, tubercle bacillus) and then for 1 minute in a solution of iodine and iodide of potassium (iodine, 1; iodide of potassium, 2; distilled water, 300). Decolorization is effected by repeated washing in alcohol. The bacteria appear on a colorless background, stained bluish-black. If it is desired, the nuclei of the cells of the tissues may be stained with some contrasting aniline color, as Bismarck brown or eosin.

The Staining of Tubercle Bacilli

1. *Ehrlich's method.* As described above, a dry specimen is prepared from the sputum. The particle selected should be taken from a purulent or cheesy part of the sputum. An easy way to find such particles is to pour the sputum upon a blackened plate or a piece of smoked glass.

The staining fluid is aniline-gentian-violet, and is prepared as follows: a saturated solution of aniline oil is made with 10 times its volume of water, and is filtered. An alcoholic solution of gentian-violet is added drop by drop to a watch-glass full of the clear aniline solution until a shining membrane appears on the surface.

The specimen, prepared side down, is placed in this solution and warmed for 10 minutes over a flame. The cover glass is then removed, washed with water and then in a 25 per cent. diluted nitric acid solution until it is colorless. Owing to its tenacity for aniline dyes, the tubercle bacillus alone is now stained, the other bacteria present having given up their coloring to the acid. To stain the tissues, the cover glass is laid in a solution of Bismarck brown for from 2 to 3 minutes. It is again washed in water and dried.

The tubercle bacilli are stained violet, the nuclei brown.

Instead of gentian-violet, one may add an alcoholic solution of fuchsin to the aniline solution, and the nuclei may be stained with methylene blue or malachite. In this event, the bacilli will be red, the nuclei blue or green.

2. *Fränkel-Gabbet's rapid method.* The following solutions must be prepared:—

A. Fuchsin, 1
Alcohol, 10
Carbolic acid, 5
Distilled water, 100

B. Methylene blue, 2
Sulphuric acid, 25
Distilled water, 100

The prepared cover glass remains 10 minutes in solution A, is washed in water, dried, and is placed for 5 minutes in solution B. It is again washed in water and dried. The specimen should now have a light blue color. If it is still red in parts, it should be put into solution B for from 1 to 3 minutes, washed, and dried. It is then mounted in oil of cloves or Canada balsam and examined. The tubercle bacilli are red; everything else in the field is blue. This method is to be recommended for its accuracy and its clear results.

The Bacteria of Diagnostic Importance

The bacteria of pus.—*a. Staphylococcus*; is arranged in irregular masses; is stained by all the aniline dyes. When a pure culture shows a growth of yellow colonies, it is known as *Staphylococcus pyogenes aureus*; when white colonies are formed in pure culture, the germ is given the name of *Staphylococcus pyogenes albus*. It may appear in any suppurative process, abscesses, phlegmona, purulent inflammations of the serous membranes, otitis, osteomyelitis, purulent inflammatory conditions following typhoid fever, etc.

b. Streptococcus; is arranged in chains and occurs in many purulent processes. The inflammatory conditions provoked by the streptococcus are more intense than those of the staphylococcus, and have a tendency to make deeper inroads upon the system.

Streptococci are the etiological elements of erysipelas; they, as well as the staphylococci, produce the various

forms of sepsis, particularly *puerperal sepsis*. They also cause endocarditis, croupous pneumonia, etc.

At present, the different varieties of streptococci are regarded as morphologically identical, differing only in the degree of virulence they manifest. The virulence of a species is determined by animal experimentation.

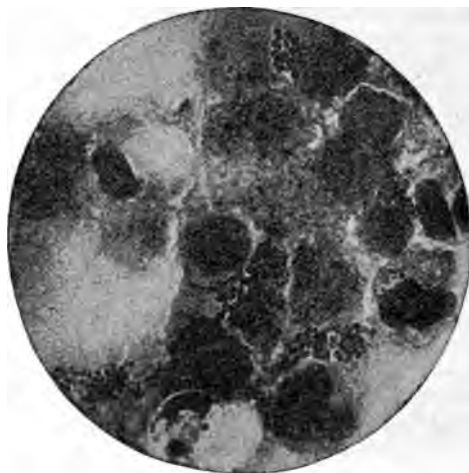


FIG. 53. — STAPHYLOCOCCI.

Gonococci (*Neisser*) (Fig. 55) are diplococci arranged like pairs of coffee-beans. They often completely fill the protoplasm of pus-cells, leaving only the nucleus free. Apparently, they are to be found only in the pus of gonorrhœa or of gonorrhœal infection (gonorrhœal conjunctivitis, cystitis, gonitis, pleurisy, and endocarditis). The presence of gonococci is accepted by most specialists as a proof of the correctness of diagnosis in doubtful cases of urethritis and leucorrhœa.

Meningococci (*Diplococci intracellulares*) (*Weichslebaum-Jäger*) are diplococci shaped like a roll, chiefly lying within

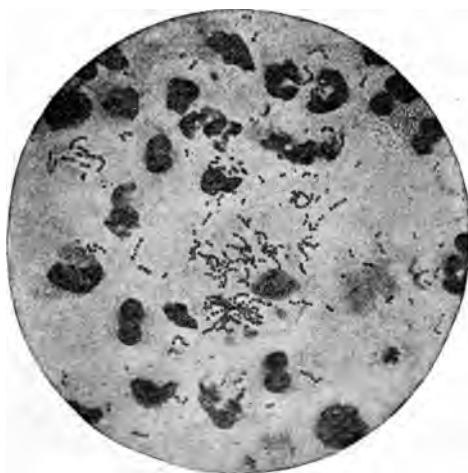


FIG. 54. — STREPTOCOCCI.

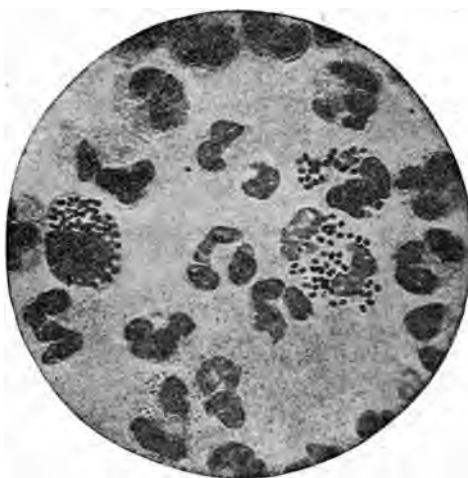


FIG. 55. — GONOCOCCI.

the cells, resembling gonococci, abundantly demonstrable during life in cases of cerebro-spinal meningitis by means of lumbar puncture.

Pneumococci, the diplococcus of pneumonia (A. Fränkel) (Fig. 56), are lancet-shaped diplococci which are regularly found in the fibrinous exudate of lungs affected with pneumonia and in the sputum accompanying pneumonia. The microscopic examination usually suffices to recognize the pneumococcus with certainty; the diagnosis is rendered more certain, however, by making a culture, and by the inoculation of the germ into rabbits which die with a typical septicæmia. The absence of the pneumococcus in the sputum argues against a diagnosis of pneumonia; its presence does not make the diagnosis absolutely certain, since it is also found in the sputum of healthy individuals. The presence of the pneumococcus in the pus of empyema makes the etiology of this disease dependent upon a former pneumonia. The pneumococcus may be responsible for purulent processes in other parts of the body (meningitis, otitis, salpingitis, etc.).

Typhoid bacilli (*Eberth*) (Fig. 57) are short rods with curved ends. They are found in patients suffering from typhoid fever in the characteristic intestinal ulcers, in the mesenteric glands, in the spleen, and, in severe cases, they may be found in other organs as well as in abscesses developing late in the disease. From the time of the beginning of the separation of the crust of the intestinal ulcer, typhoid bacilli may be found in the stools. The ordinary cover-glass preparation is not characteristic, however, and even the usual culture methods are unsatisfactory, because the bacterium coli is almost identical in culture with the typhoid bacillus.

Typhoid bacilli and the bacterium coli grow upon the culture medium—suggested by *Elsner*—of *potassium iodide*-potato-gelatine, in differential forms, while the other bacteria of the fæces do not develop thereon. By *Elsner's* method, the diagnosis of typhoid

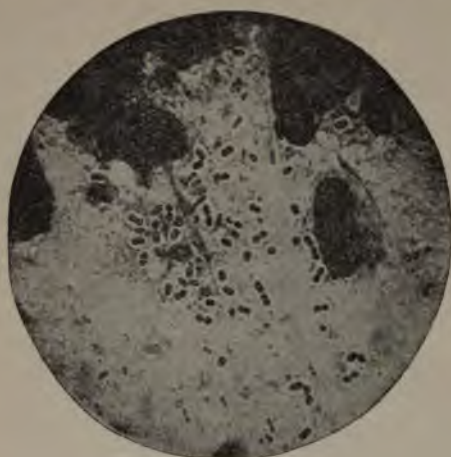


FIG. 56. — PNEUMOCOCCI.



FIG. 57. — PURE CULTURE OF TYPHOID BACILLI.

fever may often be established by the bacteriological examination of the fæces in from 24 to 48 hours.

Very recently it has been shown that the blood-serum of animals which have been immunized against typhoid bacilli possesses the specific property of affecting typhoid bacilli in such a manner that their motion is inhibited, that they resolve themselves into clumps, and become finally disintegrated (agglutination, *Pfeiffer, Gruber*). The blood-serum of persons suffering from typhoid fever has the same property (*Widal*). *Widal's reaction* for the diagnosis of typhoid fever consists in the fact that when a drop of blood-serum taken from a typhoid patient is mixed with a small quantity of a bouillion culture of the typhoid bacillus, placed in a hanging drop under the microscope, and then observed, agglutination occurs. The same reaction may be obtained when a drop of such blood is added to a 24-hour old bouillion culture of this bacillus in a test-tube, and the same placed for 12 hours in a thermostat. It is then examined for agglutination. However, the proportion of serum to bouillion culture must always be less than 1 to 25. Up to the present time, it would appear as though a positive reaction occurs only in typhoid fever.

Bacterium coli commune, short rods, scarcely to be distinguished from typhoid bacilli on a cover-glass preparation or in the ordinary culture, is found in abundance in the contents of the large intestine of man. It may produce any inflammatory or purulent condition which appears in the neighborhood of the intestines or the genito-urinary tract (peritonitis, appendicitis, abscess of the liver, cystitis, pyelitis), and may even lead to a general sepsis.

Cholera bacilli (*Koch*) (Fig. 58) are short, curved rods (comma bacilli, vibriones). They are found in great quantities in the stools of cholera patients. A diagnosis, however, cannot be made from these bacilli except in culture; for some similarly shaped saprophytes may also appear in the fæces.

The diagnosis of Asiatic cholera can be made certain only by the demonstration of the presence of the specific micro-organism. With a platinum needle, a whitish particle of mucus is selected from the suspected stool and is shaken in

a test-tube with melted gelatine; from this test-tube a second one, also containing melted gelatine, is inoculated with a platinum loop. Both test-tubes are emptied into Petri dishes. Upon the hardening of the gelatine, these dishes are kept at a temperature of 22° C. in the thermostat. In from 24 to 48 hours the colonies of cholera bacilli are recognizable. They liquefy the gelatine, and, by so doing, form



FIG. 58. — PURE CULTURE OF CHOLERA BACILLI

characteristic, funnel-shaped depressions. With a low magnifying power, the individual colonies may be distinguished by their weak lustre, by their arrangement, which bears a resemblance to fragments of broken glass, and by their irregular edges. The depressions in the gelatine are more clearly seen when the micrometer screw is employed.

If there are but few vibrios microscopically demonstrable in the fæces, the plate-culture method of *Schottelius-Koch* must be employed. A small quantity of fæces is placed in a nutritive culture medium of 1 per cent. peptone and $\frac{1}{2}$ per cent. sodium

chloride. In the thermostat these vibrios increase much more extensively in this medium than the other fæcal bacteria. As a result of their need for oxygen, they accumulate on the surface, where they form a film in about 12 hours. A small particle of this film is then inoculated on a gelatine plate.

In addition to the culture method, experiments on animals assist in the diagnosis. The *Pfeiffer* reaction is differentially diagnostic: cholera bacilli rapidly die in the peritoneal cavity of highly immunized guinea-pigs. The smallest quantity of the blood-serum of highly immunized animals will agglutinate cholera bacilli (*Gruber*).



FIG. 59. — BACILLI IN TUBERCULAR SPUTUM.

Tubercle bacilli (*Koch*) (Fig. 59) are narrow rods, about three-fourths of the size of a red blood-cell. Their staining reactions are characteristic (see p. 266). The presence of tubercle bacilli in an organ is an absolutely certain proof of the existence of tuberculosis there. They are found in the sputum (pulmonary tuberculosis), in the urine (tuberculosis of the genito-urinary tract), in the blood (miliary tuberculosis), in the fæces (tuberculosis of the intestine), in pus

(tuberculosis of the bones, empyema, etc.), and in the skin (lupus).

Tubercle bacilli in the stools of tubercular patients may be derived from sputum which has been swallowed, and do not indicate necessarily tuberculosis of the intestine.

In the smegma of the prepuce and labia, are many short rods which give some of the characteristic staining reactions of tubercle bacilli (*Smegma bacilli*). These must be borne in mind when there is a suspicion of urogenital tuberculosis. They lose their color in absolute alcohol in 1 minute, while tubercle bacilli retain their stains for several minutes.

The spirilla of relapsing fever (*Obermeier*) (Fig. 60) are found in the blood of patients suffering with relapsing fever. They are seen only when fever is present, and with a high magnifying power they may be seen

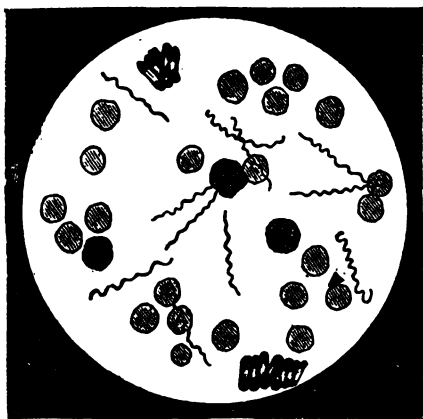


FIG. 60.—SPIRILLA FROM A CASE OF RELAPSING FEVER DURING THE FEVER.

in active movement when unstained. Cover-glass preparations may be stained with any of the aniline dyes.

Anthrax bacilli (*Davaine*) are large, thick rods, quite easily recognized in a cover-glass preparation. The diagnosis is rendered certain by inoculation upon a mouse, which dies in from 1 to 2 days after the inoculation. The blood of the mouse is found filled to overflowing with anthrax bacilli. Diagnostically, the anthrax bacillus is important because in man it is productive of large carbuncles, the specific nature of which can be recognized only by the demonstration of the bacilli.



FIG. 61. — DIPHTHERIA BACILLI.

Diphtheria bacilli (*Löffler*) (Fig. 61) are short, narrow rods, the etiological factors of diphtheritic inflammation, in which a deep necrosis of the mucous membrane goes hand in hand with the formation of a membrane. If the bacilli are not very virulent, they may call forth a simple catarrhal or fibrinous inflammation of the mucous membrane. A pure culture of diphtheria bacilli may sometimes be obtained by passing a small piece of diphtheritic membrane over several culture plates containing solidified blood-serum. In this culture medium, a shining surface develops in two days, which on microscopic examination of stained cover-glass preparations shows a collection of short rods.

In all doubtful cases of follicular angina, the membrane must be examined microscopically and by means of cultures for diphtheria bacilli. The presence of bacilli in the membranes settles the diagnosis.

Glanders bacilli (*Löffler*) may be found in profusion in the nodes of glanders; they grow on potatoes in character-

istic brown patches. If the pus of glanders or a pure culture of the bacilli is injected into the abdominal cavity of a male guinea-pig, its testicles undergo purulent inflammation (*Strauss*).

Influenza bacilli (*Pfeiffer*), very minute rods, are contained in great numbers in the bronchial mucous membrane and in pneumonic foci in influenza. Agar is a good culture medium when previously covered with fresh blood. The colonies develop as very minute drops, as clear as water and scarcely appreciable with the naked eye.

Tetanus bacilli (*Nicolaier-Kitasato*) are demonstrable in the wounds or purulent inflammations which usually evoke tetanus. The demonstration of tetanus bacilli is chiefly of theoretical interest because the clinical diagnosis is always easily established.

Actinomyces.—The ray fungus is of diagnostic importance, since it is the etiological factor of *actinomycosis*, an infectious disease running its course with chronic suppuration. The ray fungus forms, in dense masses, yellowish granules of the size of a poppy. On microscopic examination, they separate into chains of fungi with peripheral, club-shaped enlargements, resembling a bunch of grapes in their arrangement.

CHAPTER XIII

THE RÖNTGEN RAYS AS DIAGNOSTIC AIDS

IN 1895 Professor Röntgen of Würzburg discovered that, by the discharge of induced electric currents through a vacuum tube (*Crookes's*), peculiar rays of light emanated from the cathode. These X-rays, so called by Röntgen, possess the remarkable property of penetrating solid objects, and are not subject to reflection or to refraction. Looked at through a screen covered with bario-platinum cyanide, in a dark room, the X-rays evoke a bright light; upon a sensitive photographic plate they produce impressions which may be developed by ordinary daylight.

The more solid the body, the greater resistance does it offer to the passage of the Röntgen rays. Wood is more easily permeable than metal, and paper than wood. The muscles of the human body allow less easy passage of the rays than the soft parts, and the bones are least permeable.

By placing the human body between a Crookes's tube and the screen covered by the bario-platinum cyanide, the more solid parts of the body appear upon the plate as dark shadows, while the soft parts are more or less light, depending upon their respective densities. The plate thus shows a shadow-picture of the interior of the body (*actinogram*), the bony apparatus appearing very distinctly, while the organs of the chest and abdomen are easily recognized in outline.

If the body is placed between the Crookes's tube and a photographic plate enclosed in a box, the soft parts appear dark in the negative, the denser parts light. The positive

made from this shows, correspondingly, the bones, etc., dark, the soft parts more or less light.

Immediately upon the announcement of the discovery of the Röntgen rays, they began to be employed for medical purposes, and although scarcely a year and a half has elapsed since the publication of this phenomenon, it may be said that by its aid many internal diseases can be diagnosticated early or, at least, that a diagnosis can be placed beyond doubt. As the apparatus is comparatively easy to acquire and as the technic of its use is not difficult, many physicians are experimenting with the X-rays, and further advances may be awaited with certainty.

The heart. — Dilatation and pericarditis may be diagnosticated, as the size of the heart can be seen with the aid of the rays. The contractions of the heart can be distinctly witnessed upon the fluorescent screen, and this enables us to form a judgment as to the strength and rhythm of the cardiac beat which can be substantiated by the examination of the apex-beat and the pulse.

The blood-vessels. — With the aid of the Röntgen rays, calcification of the arteries can be recognized. The picture of this phenomenon on the cadaver is very striking, but it is not so sharply marked in the living subject. A more practical use can be made of the fluoroscope by observing dilatation of the arteries, by which means the diagnosis of *aneurysm of the aorta* may be made much earlier than has hitherto been the case. Aneurysm of the aorta has been seen, for example, before any local symptoms have appeared. A differential diagnosis from other mediastinal tumors can be made by the visible pulsation.

The lungs. — The lungs are visible as filling the thoracic cavity as a weak shadow, and their size can thus be easily judged. The diagnosis of emphysema can thus be made without further difficulty. Pleuritic effusions, adhesions, and large infiltrations are readily recognized by the deeper

shadows they produce. It is not yet certain if small infiltrations whose presence we have as yet no objective methods of demonstrating, can be made recognizable by means of the skiagraph.

Tumors of the lungs or of the pleuræ can be diagnosed with certainty much earlier than previously. This is, beyond doubt, an important step in the progress of diagnosis.

The abdominal organs. — The observations so far upon the abdominal organs have not been very satisfactory. Large tumors have been easily seen, but an early diagnosis of them has not yet been accomplished. It is frequently impossible to differentiate the outlines of the various organs. The *œsophagus* and *stomach* can be more readily distinguished by the preliminary passing of a stomach tube surrounded by a spiral of metal or filled with small shot. Even this measure has not furthered diagnosis. It is not yet established with certainty to what extent *renal*, *vesical*, or *biliary calculi* can be demonstrated. The greatest progress has been made in the use of this new photography in the diagnosis of diseases of the **bones** and **joints**, and in demonstrating the presence of foreign bodies. In these instances, surgery has been a greater gainer than medicine. For internal medicine, the presence of *caries of the vertebræ*, the bony changes of *rhachitis*, the anchylosed joints and the deformed epiphyses of chronic articular rheumatism, are of importance. It would seem that the Röntgen rays will offer a means of making a differential diagnosis between *gout* and *arthritis deformans*, so frequently difficult to determine.

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